Behavioural and ERP Correlates of Hypervigilance and Inhibitory Control in Spider Fear

Monique Williams

A report submitted as a partial requirement for the degree of Bachelor of Arts with Honours in Psychology at the University of Tasmania, 2016

Statement of Sources

I declare that this report is my own original work and that contributions of others have been duly acknowledged

_____ Date:_____

Monique Williams

Acknowledgements

First and foremost, I would like to express my upmost gratitude to my supervisor Dr Allison Matthews whose mentorship has been extensive. I thank you greatly for your incredible levels of patience, flexibility and endless guidance and support. My sincere thanks to Professor Kim Felmingham, who generously shared her knowledge, experience and advice. A special thank you to Val Ranson for offering her critical thinking and support, and to Amber Johnstone who generously provided her assistance in the lab. To my research partner Tess with whom I share many laughs, pizzas and late nights in the lab with, your energetic company has been uplifting and your patience and initiative throughout the year has not gone unnoticed. To the other students of the cog neuro lab, I have appreciated your support, encouragement and friendship throughout the year. A particular thank you to my partner Winston for never ceasing to provide reassurance and humour, to my mother and stepfather for their endless encouragement, and to my father whose patience and ongoing support will not be forgotten. Finally, to the participants who took part in this investigation, I thank you for your time and effort. It has made it all possible.

Table of Contents

List of Tables and Figures

Behavioural and ERP Correlates of Hypervigilance and Inhibitory Control in Spider Fear

Monique Williams

Word Count: 9958

Abstract

Attentional biases to threat are thought to arise from anxiety promoting automatic attentional processing in the form of hypervigilance, while disrupting voluntary attentional processes such as inhibitory control. There is little support for the latter in specific fear. The current study examined behavioural (RT and accuracy) and electrophysiological correlates of hypervigilance (P1 amplitude) and inhibitory control (N2 amplitude) in 15 high and 15 low spider fear females aged 18-40 years using a modified flanker go nogo task with increased cognitive load. Spider or flower go targets were flanked by incongruent images or neutral dashes. A central mushroom appeared on nogo trials, and could be flanked by either spider or flowers. High fears did not show faster RTs and greater P1 amplitude in response to spider targets as hypothesised, with both groups showing greater RTs to spider targets, and low fears demonstrating reduced P1 amplitude in the left hemisphere. Contrary to predictions, there was no fear-specific behavioural interference or reduced nogo-N2 amplitude in high fears, but they did show increased N2 amplitude on trials with spider flankers. This may suggest a compensatory inhibitory mechanism in response to feared stimuli. However, the current paradigm may not have adequately elicited automatic attentional processing or fear responses.

Attentional bias to threat occurs when selective attention is preferentially allocated to threatening compared to neutral stimuli (Bar-Haim, Lamy, Pergamin, Bakermans Kranenburg, & van IJzendoorn, 2007; Cisler & Koster, 2010). This bias has been consistently observed in individuals with high anxiety levels, and in a range of clinical anxiety disorders (Bar-Haim et al., 2007). While thought to be involved in the etiology and maintenance of clinical anxiety (Mathews & MacLeod, 2002; Van Bockstaele et al., 2014), the exact mechanisms underlying attentional bias to threat remain unclear (Cisler & Koster, 2010). The identification of such mechanisms is useful for further development of clinical treatment interventions (Van Bockstaele et al., 2014), which is of particular importance given high rates of relapse after treatment in anxiety disorders (Boschen, Neumann, & Waters, 2009).

One prominent theory proposes that attentional biases to threat arise due to anxiety disrupting the balance between two attentional systems (Eysenck, Derakshan, Santos, & Calvo, 2007). Specifically, Attentional Control Theory (ACT) argues that anxiety facilitates attention to threat by promoting automatic, bottom-up processing associated with a posterior, stimulus-driven attentional network, while simultaneously decreasing voluntary, top-down control of attention (influenced by knowledge and expectations) associated with an anterior, goal-directed attentional network (Eysenck et al., 2007). ACT is based almost exclusively on research involving high trait anxiety participants, and currently there is a lack of clear evidence for this prediction in specific fear/phobia, a distinct anxiety subtype. There is evidence of increased automatic attentional processing of threat relative to neutral stimuli in specific fear, a process referred to as *specific hypervigilance*. However, the role of voluntary control processes and their interaction with automatic processing is much less understood. Therefore, in the present study, behavioural and

electrophysiological correlates of hypervigilance and inhibitory control were assessed in spider fear using a hybrid flanker go-nogo task.

Attentional Networks Model

The attention network model (Posner & Petersen, 2012) offers a framework that distinguishes automatic and voluntary processes of attention. This model posits that an alerting, orienting and executive control network operate as anatomically and functionally distinct attentional systems. Furthermore, components of the orienting and executive control networks are synonymous with the posterior (stimulus-driven) and anterior (goal-directed) attentional systems referred to in ACT (Eysenck, Derakshan, Santos, & Calvo, 2007).

The orienting network engages prioritised processing of sensory input through orienting the 'spotlight' of attention from one location (or object) to another (Callejas, Lupianez, Funes, & Tudela, 2005; Derryberry & Reed, 2002). A dorsal fronto-parietal system within the orienting network is thought to exert rapid top-down control over attention, allowing swift covert orienting of attention (Petersen & Posner, 2012). In contrast, a ventral fronto-parietal system (including the temporo-parietal junction) is thought to be reactive to bottom-up signals arriving at the visual system, allowing rapid, stimulus-driven responses (Petersen & Posner, 2012).

The executive control network, or anterior system, directs a number of voluntary attentional processes that integrate higher order functions (Petersen & Posner, 2012). Such processes include detecting erroneous responses, monitoring and dealing with conflict, and inhibiting dominant responses (Derryberry & Reed, 2002; Petersen & Posner, 2012). Anterior regions of the frontal cortex involved in top-down control are implicated in this network, particularly the prefrontal cortex and anterior cingulate cortex (ACC) (Petersen & Posner, 2012). Recently, a dual

network view has been proposed where a cingulo-opercular and a frontoparietal system, both consisting of frontal and parietal areas, are involved in the executive control network (Petersen & Posner, 2012). The executive control network is proposed to have an interactive relationship with the orienting network, whereby it can regulate the allocation of attention via voluntary control (Derryberry & Reed, 2002). Inversely, the orienting network can exert strong influence in directing attention, which can override attentional regulation by the executive control network (Fan et al., 2009).

Attentional Bias in Spider Fear

High fear and clinical phobia of spiders is of high prevalence (Oosterink, de Jongh, & Hoogstraten, 2009). Thus, there has been much investigation into attentional biases in this subtype of fear/phobia. Behavioural evidence of enhanced automatic and reduced voluntary attentional processing of feared stimuli has been sought through the use of 'facilitation' and 'interference' paradigms, respectively.

Facilitation Effects and Hypervigilance. Facilitation effects are measured through enhanced behavioural performance in response to spider stimuli, and are interpreted to index facilitated attention to fear-relevant information. For example, in an object identification task, Kolassa, Musial, Mohr, Trippe, and Miltner (2005) found that people with spider phobia, relative to social phobia and control participants, were faster to identify images of spiders than birds or flowers. Comparable effects have been identified in dot probe tasks where participants must indicate whether a dot appears on the right or left side of a screen after it replaces either a threat related or neutral image. High relative to low spider fear participants have shown faster responses to dots replaced by spider compared to neutral and non-feared threat stimuli (Lipp & Derakshan, 2005; Mogg & Bradley, 2006). These

findings are interpreted as evidence of specific hypervigilance (Kolassa et al., 2005), or the preferential allocation and narrowing of attention towards threat compared to neutral stimuli (Eysenck, 1992). Eysenck distinguishes this from general hypervigilance, which is the propensity to attend to or become distracted by any task-irrelevant stimulus. This has also been demonstrated in spider phobia participants, where they have responded more quickly to images of both flowers and spiders compared to control and social phobia groups (Kolassa, Musial, Kolassa, & Miltner, 2006). According to Michalowski et al. (2009), in fearful individuals, exposure to phobia-relevant cues could prompt a state of general hypervigilance in response to all visual information, regardless of emotional relevance.

Hypervigilance to threat has been linked to structures involved in emotional and early visual processing. The amygdala has been implicated in automatic processing of fear and in the detection of threat (Anderson, Christoff, Panitz, De Rosa, & Gabrieli, 2003; Janak & Tye, 2015; Vuilleumier, Armony, Driver, & Dolan, 2001). Furthermore, increased activity of the amygdala has been found for high spider fear participants in response to both spider and threat-related images relative to neutral images at subliminal exposure durations (Carlsson et al., 2004). Bishop (2007) proposed an amygdala prefrontal circuitry where hyperactivity of the amygdala in response to threat, and under-recruitment of the prefrontal cortex in regulating this, facilitates enhanced projections to the visual cortex, biasing attention towards threat. In line with this, increased amygdala and visual cortex activity have been found to be correlated during viewing of faces depicting fear (Morris et al., 1998). Bishop's account is relatively concordant with ACT which alludes to increased activation of an orienting network and decreased activation of an executive control network (Eysenck, Derakshan, Santos, & Calvo, 2007). Thus,

hypervigilance to threat in specific fear could reflect increased influence of the bottom-up component of the orienting network characterised by rapid and automatic shifting of attention, (Petersen & Posner, 2012), as facilitated by amygdala hyperactivity.

Interference Effects and Inhibitory Control. Interference paradigms have also been used to examine evidence of attentional bias to threat in spider fear, by assessing the extent to which feared but task-irrelevant stimuli affect responses to neutral task-relevant stimuli. In visual search tasks, high spider fear participants demonstrate slowed identification of neutral images when spider distractors are present, compared to when only neutral or non-feared threat distractors are present (Gerdes, Alpers, & Pauli, 2008; Lipp & Waters, 2007; Rinck, Reinecke, Ellwart, Heuer, & Becker, 2005). Additionally, in emotional Stroop tasks, spider fear participants show slowed colour-naming latencies for spider-related relative to neutral words, suggesting interference where fear-relevant words are harder to ignore (Kwakkenbos, Becker, & Rinck, 2010; Thorpe & Salkovskis, 1997; Watt, McKenna, Sharrock, & Trezise, 1986). Others have failed to replicate such emotional Stroop interference in spider phobia using either schematic (Kolassa, Musial, Kolassa, & Milter, 2006) or real spider images (Kolassa, Musial, Mohr, Trippe, & Miltner, 2005). This may be due to insufficient cognitive load to elicit interference. While previous stoop tasks with spider words each used five different colours, those using pictures only used two. Thus, further investigation is needed to determine the conditions under which interference manifests in specific fear.

Interference effects are thought to result from anxiety impairing attentional control; the top-down capacity to regulate the allocation of attention and inhibit bottom-up attentional influences (Cisler & Koster, 2010; Eysenck, Derakshan,

Santos, & Calvo, 2007). Bishop's (2007) neurocognitive account proposes that hypo-activity of frontal regions plays an underlying role in this. FMRI evidence of this has been shown through decreased activity in ACC with increased levels of anxiety, and reduced activity in lateral prefrontal regions with greater expectancy of threat-related distractors (Bishop, Duncan, Brett, & Lawrence, 2004). Again, this is concordant with ACT, which posits that anxiety promotes interference from threat-related stimuli through lowering the influence of top-down processes supported by an anterior attentional network (Eysenck et al., 2007).

According to ACT, inhibitory control is a major component involved in attentional control (Eysenck, Derakshan, Santos, & Calvo, 2007). Inhibitory control has been identified as a critical lower level central executive function related to the executive control network (Miyake et al., 2000; Petersen & Posner, 2012). This function has been defined by two dissociable, but highly related sub-processes: response inhibition, which involves withholding prepotent or dominant behavioural responses, and interference suppression, which involves resisting interference from distractors (Brydges et al., 2012). According to ACT, both sub-processes are impaired by anxiety in the presence of threat-related distractors (Eysenck et al., 2007). However, ACT is based almost exclusively on research involving high trait anxiety participants. Additionally, support for Bishop's (2007) neurocognitive account has also been primarily derived from evidence using high trait anxious samples. Although anxiety and specific fear both involve intense negative emotional states, physiological symptoms and tension, there are distinct differences between the two (Ohman, 2008). While anxiety is enduring, anticipatory in nature, with it's source often obscure, fear is more acute, elicited in response to an identifiable stimulus, and typically lowers once it is no longer present (Ohman, 2008). To date,

research that has explicitly explored attentional control functions such as inhibitory control in specific fear/phobia is scarce. Thus, current understanding of the role of this mechanism in attentional biases for this anxiety subgroup is limited.

Flanker Go-Nogo Task

A hybrid version of a flanker go-nogo task is equipped to assess both hypervigilance and sub-processes of inhibitory control. The classic Eriksen and Eriksen (1974) flanker task requires a response to a central target (e.g., L) flanked by either congruent (e.g., LLLLL) or incongruent (JJLJJ) distractors on both sides. Typically, responses are slower for incongruent trials due to response conflict elicited by incompatible distractors (i.e., congruency effect), which requires interference suppression to overcome (Brydges et al., 2012; Fenske & Eastwood, 2003). In a hybrid go-nogo version of this task, participants are signalled whether they need to make (go trial) or withhold (nogo trial) a response (Heil, Osman, Wiegelmann, Rolke, & Hennighausen, 2000). Repeated presentation of go trials induces a prepotent tendency to respond, increasing the need for response inhibition in nogo trials (Brydges et al., 2012; Nee, Wager, & Jonides, 2007).
Activation of prefrontal cortex regions and the ACC has been found in both flanker and go-nogo tasks (Nee et al., 2007; Zhu, Zacks, & Slade, 2010). Furthermore, conflict between dominant and subdominant responses in these tasks makes them suitable to assess both automatic and voluntary attentional processes. Thus, this paradigm can be used to examine the distinct and interactive roles of inhibitory control and hypervigilance in attentional bias in specific fear.

Few studies have looked at attentional processes in spider fear using a flanker and/or go-nogo task. In a flanker task, Lavy, van den Hout, and Arntz (1993) found spider phobia participants showed hypervigilance to spider targets, but did not

demonstrate fear-related interference in response to neutral targets flanked by spider distractors at pre-treatment. However, as the images disappeared as soon as a response was made, the task was designed to capture avoidance from threat rather than interference suppression. More recently, Venettacci, Johnstone, Kirkby, and Matthews (2016) employed a flanker go-nogo task using schematic flower and spider images. Go and nogo trials were signaled by green and yellow stimulus strings respectively, and were presented with equal probability. Evidence of specific hypervigilance was found as high relative to low spider fear participants had faster RTs in response to spider compared to flower targets on go trials, regardless of congruency. However, high compared to low fears did not demonstrate greater interference effects in go trials in response to flower targets flanked by spiders. Venettacci et al. speculated that the hypervigilance found in high spider fear may have had a compensatory effect, enabling feared distractors to be efficiently processed and filtered out. Other findings have indicated reduced congruency effects for neutral stimuli following fear induction (Finucane, 2011) and in response to negative emotional words (Kanske & Kotz, 2010), suggesting that emotional arousal facilitates focal attention, preventing interference. This may reflect a complex relationship where the influence of the orienting network prevents impaired performance of the executive control network (Fan et al., 2009). However, this is not concordant with the predictions of ACT (Eysenck, Derakshan, Santos, & Calvo, 2007) or empirical evidence that suggests anxiety differentially impacts these two attentional networks.

An issue with the task used by Venettacci, Johnstone, Kirkby, and Matthews (2016) was that only spiders and flowers featured on incongruent trials. If slower responding was found for flower targets with spider flankers compared to spider

targets with flower flankers, it could not be determine if this was due to interference from the feared distractors in the former, or hypervigilance to the feared target in the latter. Therefore, the present study aimed to modify the task to better distinguish interference from hypervigilance.

Electrophysiological Correlates of Attention

ERPs represent averaged electrical brain activity recorded from the scalp that is time-locked to external events or stimuli (Woodman, 2010). The superior temporal resolution of this technique allows for precise measurement of swift changes in brain activity associated with early attentional processes; which are unobservable with behavioural measures alone (Woodman, 2010). Of particular relevance to the present study, the occipital P1 and frontal N2 ERP components can be used to examine attentional hypervigilance and inhibitory control, respectively.

P1 ERP Component. The P1 ERP component represents a positive peak in the ERP waveform, occurring approximately 100-130 milliseconds (ms) post-stimulus (Luck, 2014). The P1 wave is greatest at occipital sites and likely originates in the extrastriate visual cortex (Luck, 2014). The P1 component reflects visual processing at an early stage and is modulated by selective attention and arousal states (Luck, 2014; Mangun, 1995). Therefore, P1 amplitude can serve as an index of automatic capture of attention towards threatening stimuli (O'Toole & Dennis, 2012) and has been suggested to reflect a cortical mechanism related to attentional hypervigilance (Hofmann, Ellard, & Siegle, 2012), consistent with rapid shifting of attention associated with the orienting attentional network (Petersen & Posner, 2012).

In line with this, high relative to low trait anxiety participants have shown greater P1 amplitude to targets presented at the same location of threatening relative

to non-threatening images in a cue-target paradigm (Li, Li, & Luo, 2005). Studies have also found enhanced P1 amplitude in spider phobia groups compared to controls in response to schematic spider and flower images (Kolassa et al., 2007; Kolassa, Musial, Kolassa, & Miltner, 2006), and pleasant, arousing, neutral, unpleasant and spider-related real images (Michalowski et al., 2009). These findings are indicative of general cortical hypervigilance. Venettacci, Johnstone, Kirkby, and Matthews (2016) were the first to find evidence of specific cortical hypervigilance in specific fear, where high relative to low spider fear participants exhibited greater P1 amplitude in response to spider targets compared to flower targets. This was accompanied by facilitated RTs for spider targets in high fear participants. This is therefore suggestive of an early preferential selective attentional process biased towards fear-relevant stimuli (Eysenck, 1992).

N2 ERP Component. The N2 component represents a negative shift in the ERP waveform which peaks 250-300 ms post-stimulus onset (Folstein & Van Petten, 2008). Folstein and Van Petten argue that a frontocentral anterior subcomponent of the N2 is associated with cognitive control, and is elicited in go-nogo paradigms. N2 amplitude is greater on nogo relative to go trials, as they require overriding a prepotent response (Jodo & Kayama, 1992). Nogo-N2 is likely generated in the right orbitofrontal cortex; a structure thought to mediate response inhibition (Falkenstein, 2006). Nogo-N2 amplitude is increased when time pressure is enhanced (Jodo & Kayama, 1992) and when nogo trials are rarer (Nieuwenhuis, Yueng, van den Wildenberg, & Ridderinkhof, 2003), both conditions that require greater inhibitory resources when a response must be withheld. Smaller N2 amplitude for high relative to low false alarm rates on nogo trials (Falkenstein, Hoormann, & Hohnsbein, 1999), and when a task response is not successfully

withheld (Schmajuk, Liotti, Busse, & Woldorff, 2006) further suggests that nogo-N2 amplitude indexes the inhibitory resources available to override a prepotent response.

Brydges et al. (2012) employed a hybrid flanker go-nogo task using congruent and incongruent arrow direction strings, with go and nogo trials distinguished by colour. Compared to incongruent conditions, nogo conditions produced more frontally distributed N2 components with greater amplitude over frontal sites and shorter latencies. This finding, and evidence that nogo relative to go conditions elicit greater N2 amplitude (Jodo & Kayama, 1992), may suggest that nogo-N2, as an index of response inhibition, provides an optimal electrophysiological measure of inhibitory control as an executive control network function.

Compared to healthy controls, decreased nogo-N2 amplitude has been found in participants with OCD (Kim, Kim, Yoo, & Kwon, 2007) and panic disorder (Thomas, Gonsalvez, & Johnstone, 2014), suggestive of impaired inhibitory processes in these clinical groups. A reduction in N2 amplitude has been observed in participants with a spider or snake phobia relative to controls in response to their feared stimulus (Miltner et al., 2005). However, this was in a passive viewing paradigm where inhibitory control processes were unlikely to be recruited. Using the flanker go-nogo paradigm described earlier, Venettacci, Johnstone, Kirkby, and Matthews (2016) were the first to look at ERP measures of inhibitory control in specific fear. Evidence of reduced inhibitory resources to inhibit a prepotent response to feared stimuli was not found as high fear participants did not show a reduction in nogo-N2 amplitude at midline frontal sites when spiders were targets, as expected. However, insufficient task difficulty may have contributed to the lack

of both behavioural and electrophysiological evidence of reduced inhibitory control in high fear participants in this study**.**

Load theory of attention argues that distractors will more likely cause interference if cognitive load is high (Lavie, Hirst, de Fockert, & Viding, 2004). Further, ACT posits that anxiety especially impairs inhibitory control in the presence of threat distractors when task demands are greater (Eysenck, Derakshan, Santos, & Calvo, 2007). Accordingly, Venettacci, Johnstone, Kirkby, and Matthews (2016) speculated the use of colour as a go/nogo signal may have been too simple to place enough cognitive demands on participants to give rise to between-group differences in inhibitory control. While colour processing occurs early in visual processing (Railo, Salminen-Vaparanta, Henriksson, Revonsuo, & Koivisto, 2012), object recognition occurs at a later stage of processing involving top-down processes (Bar, 2003), and is therefore likely to recruit more cognitive resources. Exclusive use of flower and spider targets also meant that only two objects had to be discriminated and associated with a response in working memory throughout the task. Therefore, the present study will use a modified version of the flanker go/nogo task, designed to place greater demands on the executive control network.

Rationale and Aim

In summary, attentional biases to threat have been identified in specific fear/phobia samples and implicated in the etiology and maintenance of anxiety disorders. While there is evidence for the role of automatic attentional processes facilitated by the mechanism of hypervigilance, the role of voluntary attentional control processes underpinned by inhibitory control is less clear. Given that theory and empirical evidence suggests anxiety differentially affects the orienting and executive control attentional networks, it is important to understand the unique role

of inhibitory control in specific fear and to disentangle it's dynamic relation with earlier attentional processes. To investigate this, the impact of spider fear on behavioural (RT and accuracy) and electrophysiological correlates of hypervigilance (P1 amplitude) and inhibitory control (nogo N2 amplitude) was assessed using a modified version of the flanker go-nogo task (Venettacci, Johnstone, Kirkby, & Matthews, 2016). To isolate behavioural interference from hypervigilance, only incongruent trials were used with the inclusion of trials featuring vertical dashes as neutral flankers to provide a baseline measure of performance free from fear-related stimuli. In order to increase task difficulty and thereby increase the likelihood of between-group differences in inhibitory control, flower and spider central target images were used to signal go trials, with a third central mushroom used to signal nogo trials. Additionally, nogo trials were presented with lower probability (0.33) in aim of increasing go response tendencies and the subsequent need for response inhibition.

Hypotheses

It was hypothesised that high relative to low fear participants would have faster RTs in response to fear-relevant (spider) relative to neutral (flower) targets overall, reflecting specific behavioural hypervigilance to threat. High relative to low fear participants were also expected to have greater P1 amplitude in response to spider relative to flower targets, reflecting enhanced visual processing of feared stimuli, in line with specific cortical hypervigilance.

It was hypothesised that high relative to low fear participants would have slower RTs for flower targets when flanked by spiders relative to neutral dashes, demonstrating fear-related interference. Furthermore, it was predicted that high relative to low fear participants would have reduced N2 amplitude on nogo relative

to go trials with spider relative to flower flankers present, suggesting a reduction in inhibitory resources to override a prepotent response to the distracting feared go stimuli.

Method

Participants

Participants were 30 females (15 high fear) aged 18-40 years old (*M*=23.57, *SD*=6.00). Only females were recruited to control for potential sex differences in cognitive processing, and given higher rates of specific fear and phobia in females than males (Oosterink, de Jongh, & Hoogstraten, 2009). G*Power 3.1.9.2 estimates indicated sample sizes of 15 per group were sufficient to detect moderate sized effects (*f* = 0.25) (alpha = .05, power = .9).

A total of 209 females completed the screening questionnaire. The aim was to recruit those with scores in the upper (17 or above) and lower (5 or below) quartile on the Spider Phobia Questionnaire (SPQ; Watts & Sharrock, 1984). In the final sample, high and low fear participants' scores fell in the 70th (14 or above) and 40th (7 or below) percentile, respectively. Participants were either psychology undergraduates at the University of Tasmania (UTAS) who received course credit to participate, or volunteers known to the investigators or recruited via UTAS and social media advertisement. Four (2 high fear) of 34 participants who completed the experimental session were excluded from the analysis due to accuracy rates less than 80% in at least one condition.

The exclusion criteria included a history of neurological or psychiatric disorders (other than anxiety or affective), seizure, head injury, loss of consciousness, serious physical conditions, current use of psychoactive medication (other than anti-depressants), illicit drug use within the last month, or more than 50

lifetime occasions, and pregnancy (see Appendix A). Potential alcohol dependence (scores greater than 19 on Alcohol Use Disorders Identification Test; Babor, Higgens-Biddle, Saunders, & Monteiro, 2001), and high levels of psychological distress (scores greater than 30 on Kessler Psychological Distress scale; Kessler et al., 2002) also formed part of the exclusion criteria. However, one participant had a K10 score of 33. Participants were also asked to abstain from caffeine (2 hours), tobacco (2 hours), alcohol (24 hours) and illicit drugs prior to the session. All participants were right handed, except four (2 Low fear), and one participant did not report their handedness.

Materials and Apparatus

Questionnaire Measures. The Spider Phobia Questionnaire (SPQ; Watts & Sharrock, 1984) assesses dimensions of coping\avoidance, preoccupation, and vigilance in response to spiders via 43 yes/no questions (e.g., "*do you worry more about spiders than most people*?"). Ten items relating to factual knowledge about spiders were removed for the current study. Five items are reverse scored to avoid response bias. Evidence of excellent internal consistency (Cronbach's $\alpha = 0.91$), test-retest reliability ($r = .94$), convergent validity with measures of anxiety and avoidance, and the ability to distinguish phobic from non-phobic individuals has been found for the SPQ (Muris & Merckelbach, 1996).

The Fear of Spiders Questionnaire (FSQ; Szymanski & O'Donohue, 1995) was used as a secondary measure of spider fear. Compared to the SPQ, FSQ explicitly assesses current functioning and is superior in capturing spider fear in the low fear range (Muris & Merckelbach, 1996). Eighteen items assess responsiveness to spiders (e.g., "*if I saw a spider now, I would feel very panicky*") on a 7-point Likert scale where 1 = definitely not, and 7 = absolutely. Higher scores indicate

greater intensity of phobic symptoms. The FSQ shows excellent internal consistency (Cronbach's α = 0.95-0.97), test-retest reliability (*r* = .91), and convergent validity with anxiety and avoidance measures (Muris & Merckelbach, 1996).

The State-Trait Anxiety Inventory Form Y-2 (STAI; Spielberger, 1983) includes 20 trait anxiety items. Respondents are required to evaluate how they feel generally in response to statements designed to capture worry, discomfort and stress (e.g., "*I feel nervous and restless*") on a 4-point Likert scale where 1 = almost never, and 4 = almost always. Higher scores indicate greater levels of trait anxiety. Good internal consistency (Cronbach's α = 0.90-0.91) (Spielberger, 1983) and evidence of convergent validity with other anxiety inventories (e.g., Antony, Bieling, Cox, Enns, & Swinson, 1998; Creamer, Foran, & Bell, 1995) has been found for the trait anxiety sub-scale. State anxiety was measured using the Subjective Units of Distress Scale (SUDS; Wolpe, 1969) where respondents indicate their current subjective intensity of anxiety on a scale where 0 = no anxiety, and 100 = extreme anxiety.

The Kessler Psychological Distress scale (K10; Kessler et al., 2002) comprises ten items that assess experiences of psychological distress over the last four weeks (e.g., "*did you feel nervous*?") on a 5-point Likert scale where 1 = all of the time, and 5 = none of the time. Possible scores range from 10 to 50, where a score above 30 indicates very high levels of psychological distress. The K10 has demonstrated excellent internal consistency (Cronbach's α = 0.93) (Kessler et al., 2002).

The Alcohol Use Disorders Identification Test (AUDIT; Babor, Higgins-Biddle, Saunders, & Monteiro, 2001) includes ten items that assess alcohol consumption, dependence, and related problems. Six frequency questions are rated on a 5-point scale (e.g., 'never' to 'daily or almost daily'). Two questions assessing

harm from drinking are rated on a 3-point scale from ‘no’ to ‘yes, during the last year’. Two items assess the amount of alcohol consumed in a typical session, and frequency of consuming an alcoholic drink. Evidence of both convergent and discriminative validity has been found for this scale (Bohn, Babor, & Kranzler, 1995).

The Wechsler Test of Adult Reading (WTAR; Wechsler, 2001) assesses intellectual functioning. Participants are asked to pronounce fifty irregularly spelled words. One point is awarded per correct response, with 50 being the maximum possible score. The test is ended after 12 consecutive incorrect responses. Good internal consistency (Cronbach’s $\alpha = 0.87\text{-}0.97$), test-retest reliability ($r = .90\text{-}.94$), and convergent validity with other measures of intellectual functioning has been found for this test (Wechsler, 2001).

Karolinska Sleepiness Scale (KSS; Åkerstedt & Gillberg, 1990) is a single-item measure of subjective level of sleepiness using a 9-point scale where 1 = very alert, and 9 = very sleepy, great effort to stay awake, fighting.

A Video Gaming Experience Questionnaire (VGEQ) was developed for the current study to assess frequency of video game play via one question. Response options range from 1 = never play video games, and 5 = often play video games (more than 5 hours a week). While psychometric properties for this questionnaire have not been assessed, it was administered in an attempt to test for a potential confound given evidence of enhanced visual attention skills in regular video gamers (Dye, Green, & Bavelier, 2009).

Flanker Go-NoGo Task. A hybrid flanker go-nogo task was presented via NeuroScan STIM 3.1 software. White schematic images of a spider and a flower adapted from Kolassa, Musial, Kolassa, and Miltner (2006) were used as fear-

relevant and neutral stimuli, respectively. On go trials, a target (spider or flower) was flanked by either two incongruent images (spider or flower) or two neutral dash distractors on either side. On nogo trials, the central stimulus was a white schematic mushroom flanked by either two spiders or flowers on either side. In total, six stimulus combinations were used (see Figure 1). There were 90 trials for each condition (total of 540 trials), presented in a fully randomised order with equal probability over four blocks of 135 trials. For each trial, a black screen displayed a central fixation point for 300ms followed by the five picture string (7mm height x 36mm length, subtending 0.80^{o} x 4.12^{o} of visual angle) for 250ms, with the target appearing in identical position to the preceding fixation point. The inter trial interval was varied randomly across three stimulus onset asynchronies: 1750ms, 1850ms, and 1950ms, in an attempt to minimise latency jitter.

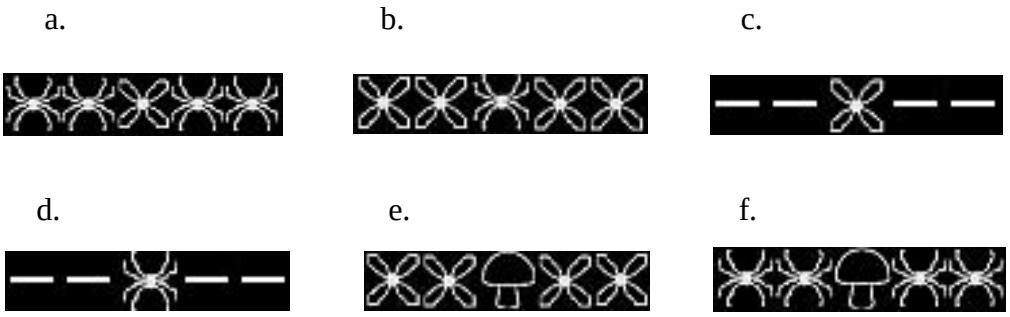

Figure 1. Stimulus strings used in the flanker go nogo task. Flower incongruent (a), spider incongruent (b), flower neutral (c), spider neutral (d) were used for go conditions. Mushroom flower-incongruent (e) and mushroom spider-incongruent (f) were used for nogo conditions.

Electrophysiological (EEG) Recording. The NeuroSCAN system (Scan 4.5 software) was used to obtain EGG recordings, using a 32-channel Quik-Cap with

Ag/AgCl sintered electrodes. EEG data was sampled continuously at a rate of 1000Hz, according to the international 10-20 system of electrode placement from 32 sites. Electrode impedance was kept below 10kΩ. Electrodes were placed on the outer canthi of both eyes and the upper and lower left eye to measure horizontal and vertical electro-oculographic (EOG) activity, and all electrodes were referenced to linked mastoids. During editing, continuous EEG data and behavioural data were merged and EEG data was filtered using a Zero-phase-shift low pass filter (30Hz, 24 dB/Oct). To reduce the impact of eye blinks on other electrode channels, ocular artefact rejection was employed. Epochs were then extracted from the data from 100ms before stimulus onset to 900ms post stimulus. Baseline correction and artefact rejection was then carried out with trials containing artefacts above 70 μV and below -70 μV rejected. The occipital P1 and frontal N2 components were defined as the maximum amplitude between 80-120ms and 230-390ms post stimulus onset respectively and were derived from grand averaged waveforms for each condition.

Procedure

The current study was approved by the University of Tasmania Human Research Ethics Committee (see Appendix B). Eligible participants attended an experimental session of approximately two hours. Upon arrival, participants were given an information sheet and provided informed consent (see Appendix C). Participants were then asked about their alcohol, caffeine, nicotine, drug and prescription medication use to ensure they were still eligible (see Appendix D). The KSS (Åkerstedt & Gillberg, 1990), STAI (Spielberger, 1983), VGEQ (see Appendix E), and WTAR (Wechsler, 2001) were then completed.

Following EEG set-up, participants were seated in front of a computer screen

at a viewing distance of approximately 50cm to complete the flanker go/nogo task followed by another task that was not part of the current study, in standardised order. Participants were asked to indicate whether the middle target was a spider or a flower via a button press on go trials, and to withhold any response on nogo trials when the middle target was a mushroom. Instructions were given to respond as quickly and accurately as possible. For the first two blocks, the right forefinger was used to respond to spiders and the left forefinger was used to respond to flowers, with this response pattern reversed for the remaining two blocks. The order of right and left finger assignment was counterbalanced between participants. Ten practice trials were completed prior to commencing the first and third block. Participants were asked to provide SUDS ratings at the beginning of each block. Breaks were given between blocks to minimise fatigue effects. The session concluded with debriefing.

Design and Data Analysis

ANOVA was chosen as the analysis for the current study, as this is the most conventional and parsimonious approach in ERP studies where a small number of electrode sites are assessed (Luck, 2014). Behavioural DVs were mean RT (ms) for go trials and mean accuracy (% of correct trials). The electrophysiological DVs were peak P1 for go trials and N2 amplitude. Analysis of the N2 component was confined to the midline frontal Fz site, as a preliminary analysis indicated greater N2 amplitude at Fz compared to Cz (p<.001) and FCz (p=.001), $F(1, 34)=33.27$, p<.001, η_p^2=.552, Greenhouse-Geisser corrected. Analysis of the P1 component was confined to sites O1 and O2 as greater P1 amplitude has previously been shown in lateral compared to central occipital sites (e.g., Kolassa et al., 2007).

Addressing hypotheses relating to behavioural and cortical hypervigilance,

and behavioural interference, RT and accuracy to go stimuli were analysed with separate 2 (Group: high fear, low fear) x 2 (Target image: spider, flower) x 2 (Flanker type: incongruent image, neutral) mixed ANOVAs, with the additional variable of Hemisphere (left, right) included for the P1 amplitude analysis. Addressing the hypothesis relating to response inhibition, separate 2 (Group: high fear, low fear) x 2 (Trial: go, nogo) x 2 (Flanker image: spider, flower) mixed ANOVAs were used to analyse accuracy and N2 amplitude.

Levene's test indicated a violation of homogeneity of variance for spider and flower incongruent trials in accuracy analyses, and SUDs ratings in blocks 3 and 4. However, ANOVA is argued to be fairly robust to violation of this assumption when sample sizes are equal, and when the largest variance does not exceed the smallest by four times (Howell, 2012), which was the case in the current analysis. Only significant ($p < .05$) interactions of theoretical relevance were further investigated via analysis of simple effects, with Bonferroni corrections applied to keep the family-wise error rate at .05. Greenhouse-Geisser corrections were applied to within-group effects with more than two levels to counter likely violations of sphericity. For omnibus ANOVAs, partial eta square represented the proportion of variance in a DV accounted for by the IVs, with effect sizes interpreted as 0.01=small, 0.06=medium, 0.14=large (Cohen, 1988). However, Cohen's *d* was used to provide a standardised measure of differences between means for pairwise comparisons, and was interpreted in accordance to Cohen's (1992) guidelines (0.2=small, 0.5=medium, 0.8=large).

Results

Demographics

Table 1 shows the mean age and raw scores for questionnaire measures for both groups. There were no significant differences between the groups on age, sleepiness on the day of testing (KSS), intellectual functioning (WTAR), video gaming experience (VGEQ), alcohol usage (AUDIT), and trait anxiety (STAI). As expected, high fear participants had significantly greater scores for both measures of spider fear (SPQ, FSQ). High fear participants also had significantly higher scores for psychological distress (K10) compared to low fear participants.

Behavioural Data

Accuracy. Cell means are presented in Table 2. For analysis of accuracy on go trials, there was a main effect of Flanker type, $F(1,28)=10.21$, $p=.003$, $\eta_p^2=.267$, with a significantly lower accuracy for incongruent image ($M=93.0$, $SD=3.4$, 95%CI[91.2,94.8) relative to neutral flanker trials ($M=94.9$, $SD=2.8$, 95%CI[93.4,96.4]). All other main effects and interactions were non-significant (ps>.05, see Table F1 Appendix F).

For analysis of accuracy on incongruent go and nogo trials, there was a main effect of Trial type, $F(1,28)=60.27$, $p<.001$, $\eta_p^2=.683$, with responses significantly more accurate for nogo trials ($M=99.2$, $SD=0.8$, 95%CI[98.8,99.6]) compared to go trials ($M=93.0$, $SD=3.4$, 95%CI[91.2,94.8]). There was also a significant main effect of Flanker image, $F(1,28)=6.17$, $p=.019$, $\eta_p^2=.180$, with lower accuracy in response to trials with spider flankers ($M=95.7$, $SD=2.2$, 95%CI[94.5,96.8]) compared to those with flower flankers ($M=96.5$, $SD=1.8$, 95%CI[95.5,97.5]). Overall, high fear participants ($M=95.1$, $SD=2.7$, 95%CI[93.6,96.5]) were less accurate than low fear

participants (*M*=97.1, *SD*=2.7, 95%CI[95.7,98.5]), *F*(1,28)=4.28, *p*=.048, η_p^2=.133. All interactions were non-significant (*p*s>.05, see Table F2, Appendix F).

Table 1

Mean Age and Raw Scores on Measures of Spider Fear, Sleepiness, Reading Ability, Video Game Usage, Alcohol Usage, Anxiety, and Psychological Distress for High and Low Spider Fear Groups

	Low fear	High fear			
Variable	*M* (*SD*)	*M* (*SD*)	*F*(1, 28)	*p*	Cohen's *d*
Age	23.3 (5.0)	23.8 (7.0)	0.04	.84	0.1
$SPQ_{/33}$	3.9 (1.8)	18.9 (3.9)	183.89	<.001	5.0
$FSQ_{/126}$	29.1 (12.8)	96.5 (13.6)	194.58	<.001	5.1
$KSS_{/9}$	3.5 (1.6)	4.3 (1.3)	2.36	.14	0.6
$WTAR_{/50}$	109.6 (11.3)	110.2 (26.9)	0.01	.94	0.03
$VGEQ_{/5}$	2.3 (1.2)	2.5 (1.5)	0.17	.68	0.2
$AUDIT_{/40}$	4.6 (2.0)	5.6 (4.6)	0.60	.45	0.3
$STAI_{/80}$	35.8 (12.4)	39.9 (7.7)	1.21	.28	0.4
$K10_{/50}$	14.9 (5.0)	19.5 (6.1)	5.09	.03	0.8

Table 2

Mean Accuracy (% correct) with SD in Parentheses and 95% Confidence Intervals for High and Low Fear Groups for each Stimulus Condition

	Low Fear		High Fear	
	M (*SD*)	95% CI	*M* (*SD*)	95% CI
Go trials				
Flower Incongruent	93.6 (3.9)	[91, 97]	90.7 (6.7)	[88, 94]
Flower Neutral	95.9 (4.1)	[94, 98]	93.4 (4.6)	[91, 96]
Spider Incongruent	95.6 (3.2)	[93, 98]	92.0 (6.0)	[90, 95]
Spider Neutral	95.9 (4.2)	[94, 98]	94.4 (4.6)	[92, 97]
Nogo trials				
Flower-Incongruent	99.6 (0.7)	[99, 100]	98.9 (1.1)	[98, 99]
Spider-Incongruent	99.6 (0.7)	[99, 100]	98.7 (2.0)	[98, 100]

Reaction Time. Cell means are shown in Table 3. Analysis of go trials revealed a significant main effect of Target image, $F(1,28)=35.42$, $p<.001$, $\eta_p^2=.558$, with faster RT to spider (M=526.3, SD=31.6, 95%CI[509.5,543.0]) compared to flower targets (M=550.4, SD=35.7, 95%CI[531.5,569.2]). However, as shown in Figure 2, the hypothesised Group x Target image interaction was non-significant, $F(1,28)=1.00$, $p=.326$, $\eta_p^2=.034$, with both high and low fear participants showing faster RTs to spider relative to flower targets.

Table 3

Mean RT (ms) with SD in Parentheses and 95% Confidence Intervals for High and Low Fear Groups for each Stimulus Condition

	Low Fear		High Fear	
Condition	*M* (*SD*)	95% CI	*M* (*SD*)	95% CI
Flower Incongruent	556.6 (38.3)	[530, 582]	587.5 (58.5)	[561, 613]
Flower Neutral	514.6 (43.5)	[486, 542]	542.8 (61.4)	[514, 570]
Spider Incongruent	535.4 (31.8)	[513, 557]	555.2 (50.5)	[532, 577]
Spider Neutral	495.8 (34.8)	[469, 522]	518.9 (62.2)	[492, 545]

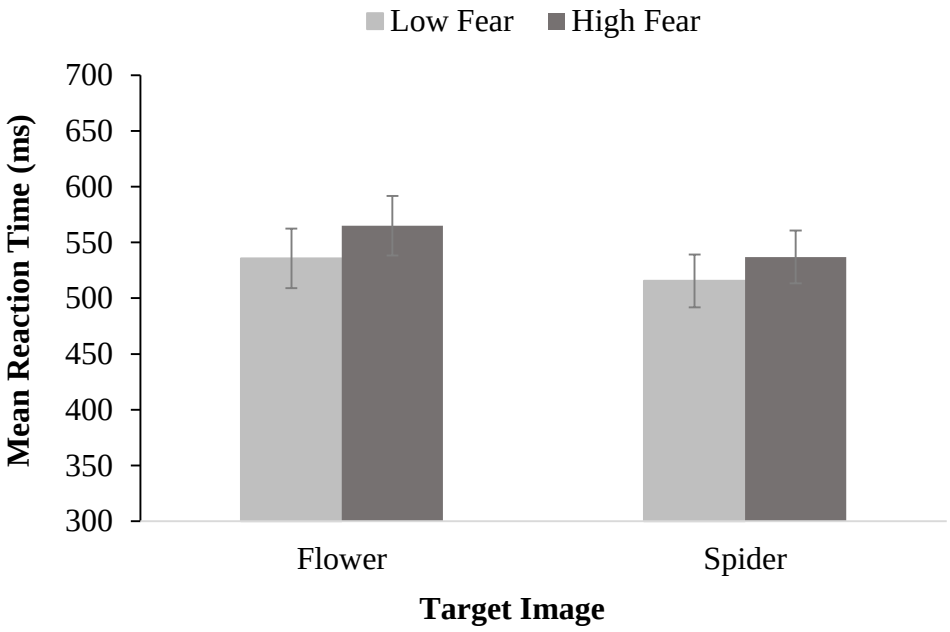

Figure 2. Mean reaction time for flower and spider targets in low and high fear groups (error bars represent 95% CIs).

Overall, responses were slower on trials with incongruent (*M*=558.6, *SD*=31.3, 95%CI[542.1,575.2]) compared to neutral flankers (*M*=518.0, *SD*=35.2, 95%CI[499.4,536.6]), $F(1,28)=187.77$, $<p.001$, $\eta_p^2=.870$. However, as shown in Figure 3, the hypothesised Group x Target image x Flanker type interaction was non-significant, $F(1,28)=0.297$, $p=.590$, $\eta_p^2=.010$, with both groups showing slower RTs to trials with incongruent image flankers, that did not significantly vary for spider and flower targets. No other main effects or interactions were significant (*p*s>.05, see Table F1 Appendix F).

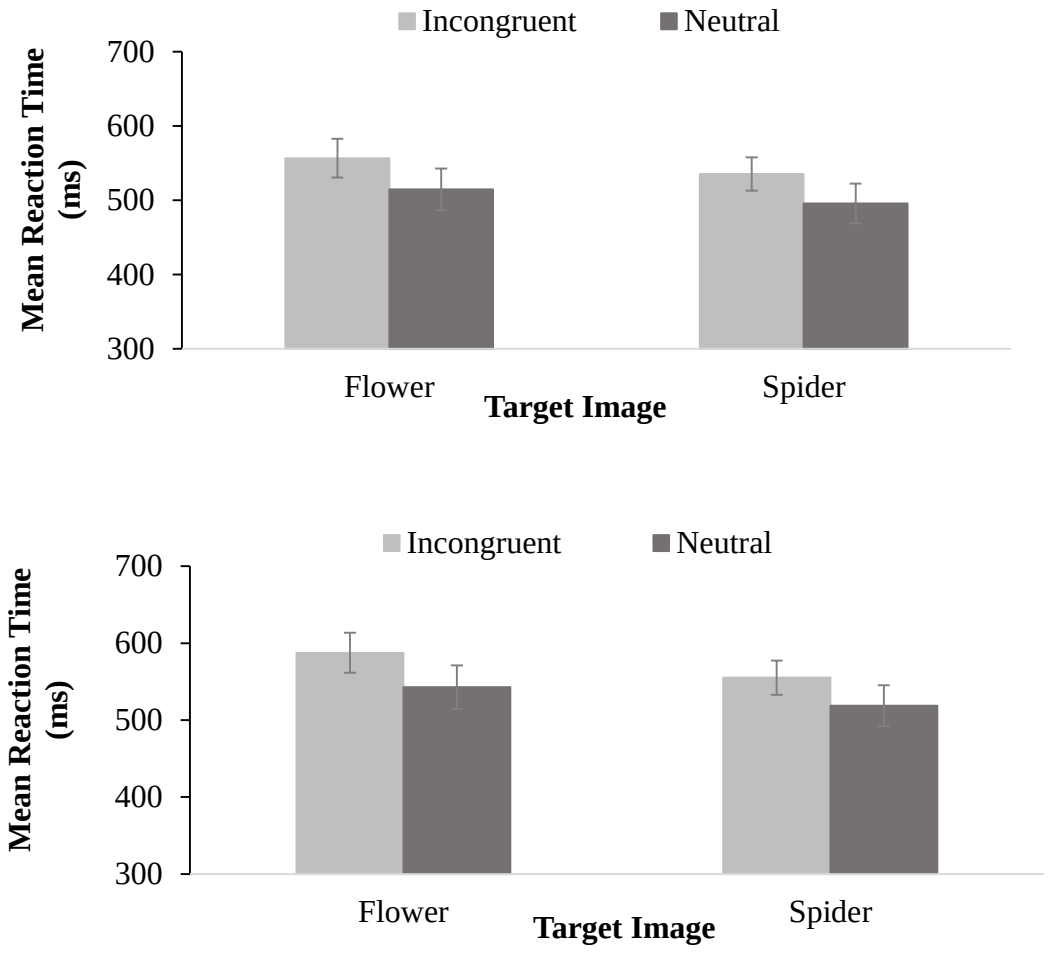

Figure 3. Mean reaction time for flower and spider targets for incongruent image and neutral flanker trials in low (top) and high (bottom) fear participants (error bars represent 95% CIs).

Electrophysiological Data

Peak P1 Amplitude. Figures 4 and 5 show the grand mean averaged waveforms in response to go trials at the left (O1) and right (O2) lateral occipital sites for low and high fear participants, respectively. Descriptive statistics for P1 amplitude are shown in Table 4.

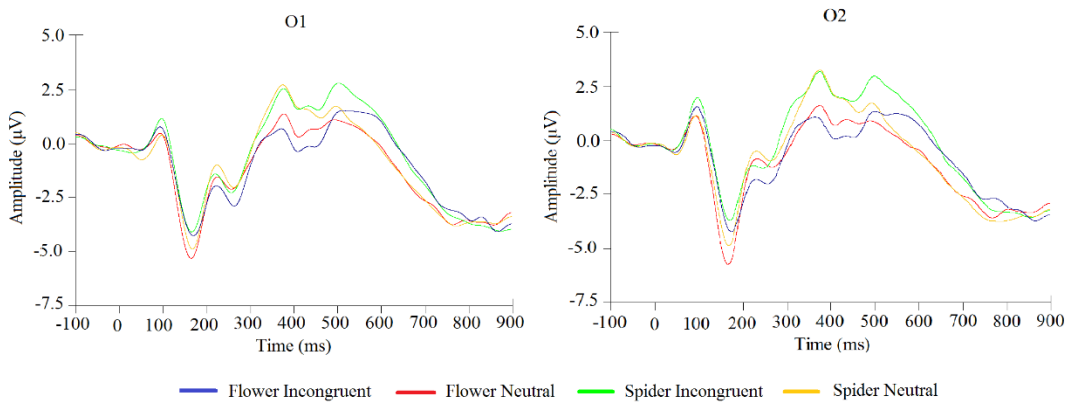

Figure 4. Grand mean averaged waveforms for low fear participants at occipital O1 (left) and O2 (right) electrode sites for go conditions.

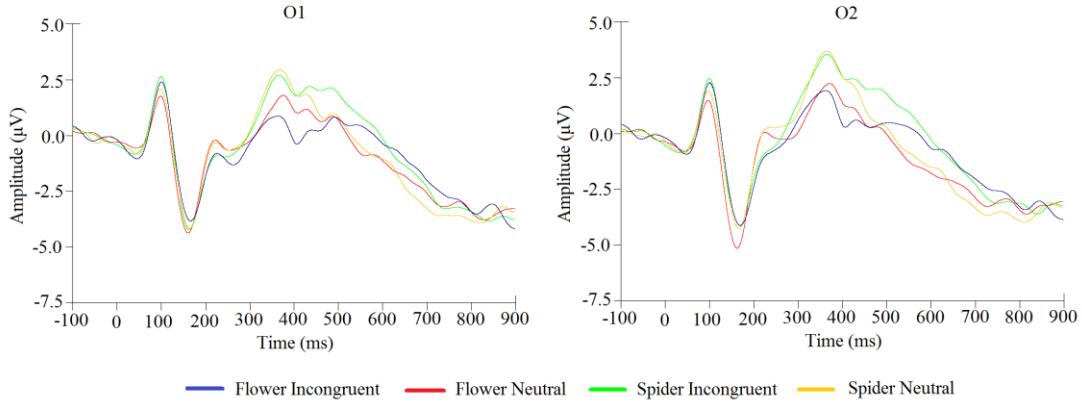

Figure 5. Grand mean averaged waveforms for high fear participants at occipital O1 (left) and O2 (right) sites for go conditions.

Table 4

Mean P1 Amplitude (μV) at Left (O1) and Right (O2) Occipital Sites with SD in Parentheses and 95% Confidence Intervals for High and Low Fear Groups for Go Stimulus Conditions

		Low Fear		High Fear	
Hemisphere	Condition	*M* (*SD*)	95% CI	*M* (*SD*)	95% CI
Left	F Incongruent	2.3 (2.5)	[0.9, 3.7]	4.1 (2.9)	[2.7, 5.6]
	F Neutral	1.7 (1.6)	[0.6, 2.7]	3.1 (2.3)	[2.0, 4.1]
	S Incongruent	2.5 (2.4)	[1.2, 3.9]	4.3 (2.7)	[2.9, 5.7]
	S Neutral	1.5 (1.8)	[0.4, 2.6]	3.4 (2.3)	[2.3, 4.5]
Right	F Incongruent	3.5 (2.7)	[2.2, 4.8]	3.7 (2.3)	[2.4, 5.0]
	F Neutral	2.5 (1.8)	[1.6, 3.3]	2.7 (1.3)	[1.9, 3.6]
	S Incongruent	3.6 (2.6)	[2.4, 4.8]	3.8 (1.8)	[2.6, 5.0]
	S Neutral	2.6 (1.9)	[1.7, 3.5]	3.0 (1.4)	[2.1, 3.9]

Note: S=spider, F=flower

There were non-significant main effects for both Group, $F(1,28)=2.24$, $p=.146$, $\eta_p^2=.074$, and Target image, $F(1,28)=2.78$, $p=.106$, $\eta_p^2=.090$. As Figure 6 shows, the hypothesised Group x Target image interaction was also not significant, $F(1,28)=0.62$, $p=.440$, $\eta_p^2=.021$, with both groups demonstrating a non-significant difference in P1 amplitude between spider and flower targets.

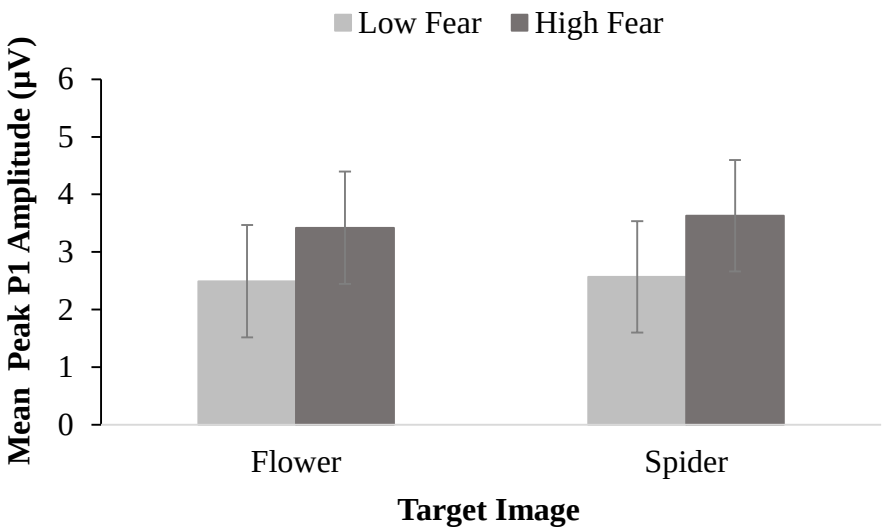

Figure 6. Mean peak P1 amplitude for flower and spider targets in low and high fear groups (error bars represent 95% CIs).

However, there was a significant Group x Hemisphere interaction (see Figure 7), $F(1,28)=4.47$, $p=.043$, $\eta_p^2=.138$. Simple main effects of Hemisphere were analysed separately at each level of Group ($\alpha=.025$, Bonferroni corrected). There was a simple main effect of Hemisphere for low fear participants, $F(1,14)=10.22$, $p=.006$, with significantly greater P1 amplitude in the right relative to left hemisphere, with this effect moderate in magnitude ($d=0.50$). In contrast, high fear participants did not show a significant difference in P1 amplitude in the right compared to the left hemisphere, $F(1,14)=0.46$, $p=.510$, $d=0.19$. Simple main effects of Group were also analysed at each level of Hemisphere ($\alpha=.025$, Bonferroni corrected). While there was a trend towards significantly greater P1 amplitude for high relative to low fear participants in the left hemisphere, $F(1,28)=4.53$, $p=.042$, $d=0.78$, there were no between-group differences in the right hemisphere, $F(1,28)=0.16$, $p=.693$, $d=0.15$.

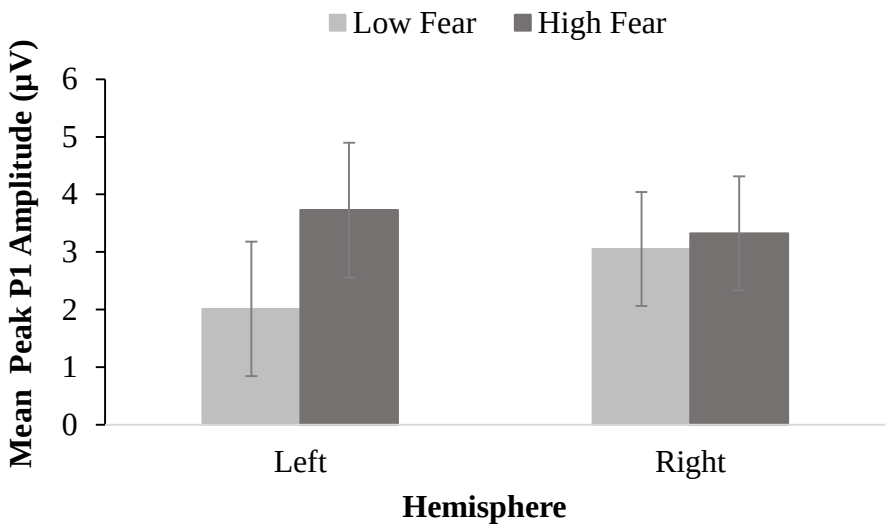

Figure 7. Mean peak P1 amplitude for low and high fear groups at occipital sites O1 and O2 (error bars represent 95% CIs).

The main effect of Flanker type was significant, P1 amplitude greater in response to incongruent (*M*=3.5, *SD*=1.5, 95%CI[2.7,4.3]) compared to neutral flanker trials (*M*=2.6, *SD*=1.1, 95%CI[2.0,3.2]), $F(1,28)=21.96$, $p<.001$, $\eta_p^2=.440$. No other main effects or interactions were significant (*p*s<.05, see Table F1 Appendix F).

Peak N2 Amplitude. Figure 8 shows the grand mean averaged waveforms at the midline frontal site (Fz) for high (left) and low (right) fear participants in response to incongruent go and nogo conditions. Inspection of the Figure suggests peak N2 amplitude (peaking at approx. 300-320ms) was greater for nogo trials compared to go trials in both groups. Descriptive statistics are shown in Table 5.

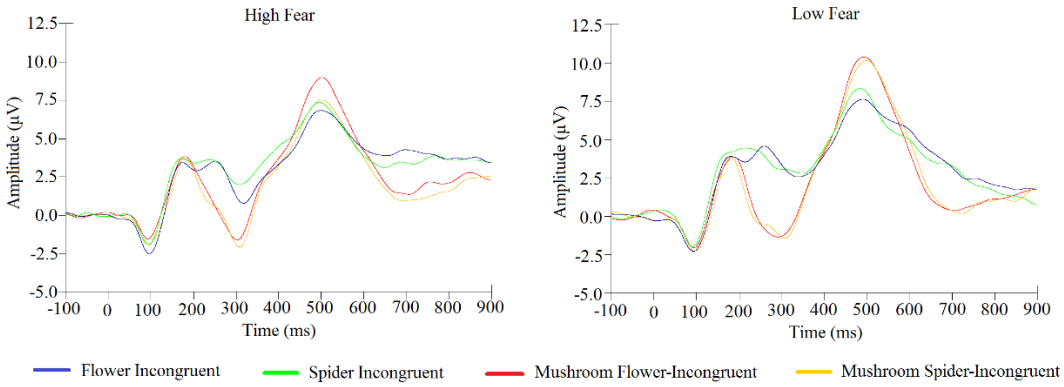

Figure 8. Grand mean averaged waveforms for high (left) and low (right) fear groups at the midline frontal electrode site (Fz) for go incongruent image (flower, spider) and nogo (mushroom) conditions.

Table 5

Mean N2 Amplitude (μV) with SD in Parentheses and 95% Confidence Intervals for High and Low Fear Groups for Incongruent Go and Nogo Stimulus Conditions

	Low Fear		High Fear	
	M (*SD*)	95% CI	*M* (*SD*)	95% CI
Go trials				
Flower Incongruent	0.3 (5.0)	[-2.0, 2.6]	-1.3 (3.4)	[-3.5, 1.0]
Spider Incongruent	-0.2 (4.5)	[-2.4, 2.0]	0.3 (3.7)	[-1.9, 2.5]
Nogo trials				
Spider-Incongruent	-3.4 (4.1)	[-5.3, -1.4]	-3.4 (3.4)	[-5.4, -1.5]
Flower-Incongruent	-3.6 (3.9)	[-5.6, -1.5]	-3.2 (3.8)	[-5.2, -1.1]

A main effect of Trial type revealed that N2 amplitude was greater on nogo (*M*=-3.4, *SD*=3.8, 95%CI[-4.8,-2.0]) compared to go trials (*M*=-0.2, *SD*=3.9, 95%CI[-1.7,1.3]), $F(1,28)=80.30$, $p<.001$, $\eta_p^2=.741$. There was a trend for a Group x Flanker image interaction (see Figure 9), $F(1,28)=3.90$, $p=.058$, $\eta_p^2=.122$. Bonferroni corrected ($\alpha=.025$) tests of simple main effects of Flanker image were conducted for each group. High fear participants had significantly greater N2 amplitude on trials with spider (*M*=-2.3, *SD*=3.1, 95%CI[-4.1,-0.6]) relative to flower flankers (*M*=-1.5, *SD*=3.5, 95%CI[-3.4,0.5]), $F(1,14)=7.62$, $p=.015$, $d=0.27$, with a small effect size noted. Low fear participants did not show a significant difference in N2 amplitude in response to spider (*M*=-1.5, *SD*=4.4, 95%CI[-4.0,0.9]) and flower flankers (*M*=-1.9, *SD*=4.2, 95%CI[-4.2,0.4]), $F(1,14)=0.43$, $p=.522$, $d=0.08$, with a negligible effect noted. Simple main effects of Group for each Flanker image ($\alpha=.025$, Bonferroni corrected) High and low fear groups did not significantly differ in N2 amplitude in response to trials with spider, $F(1, 28)=0.35$, $p=.562$, $d=0.21$, or flower, $F(1, 28)=0.10$, $p=.755$, $d=0.12$, flankers.

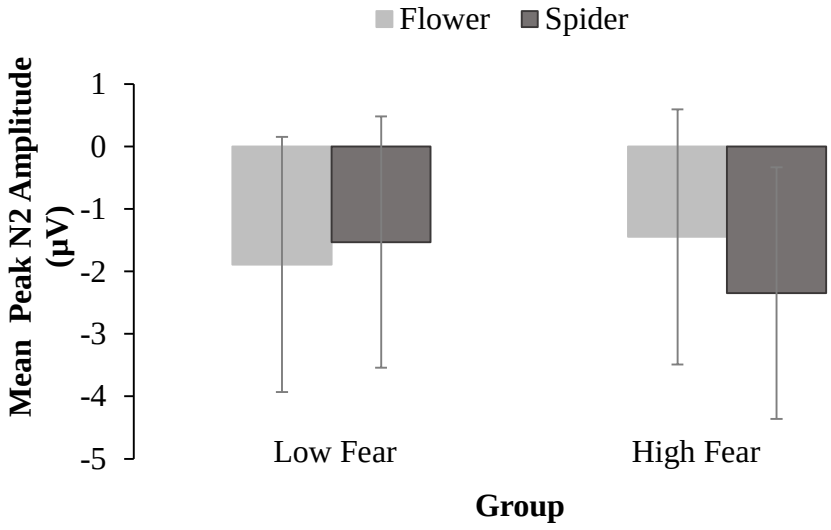

Figure 9. Mean peak N2 amplitude for low and high fear participants at frontal site Fz for trials with flower and spider flankers (error bars represent 95% CIs).

The hypothesised Group x Trial type x Flanker image interaction was non-significant (see Figure 10), $F(1,28)=2.20$, $p=.150$, $\eta_p^2=.073$, with the greater N2 amplitude in the high fear group for spider flankers not significantly differing according to Trial type. No other significant main effects or interactions were found ($ps<.05$, see Table F2 Appendix F).

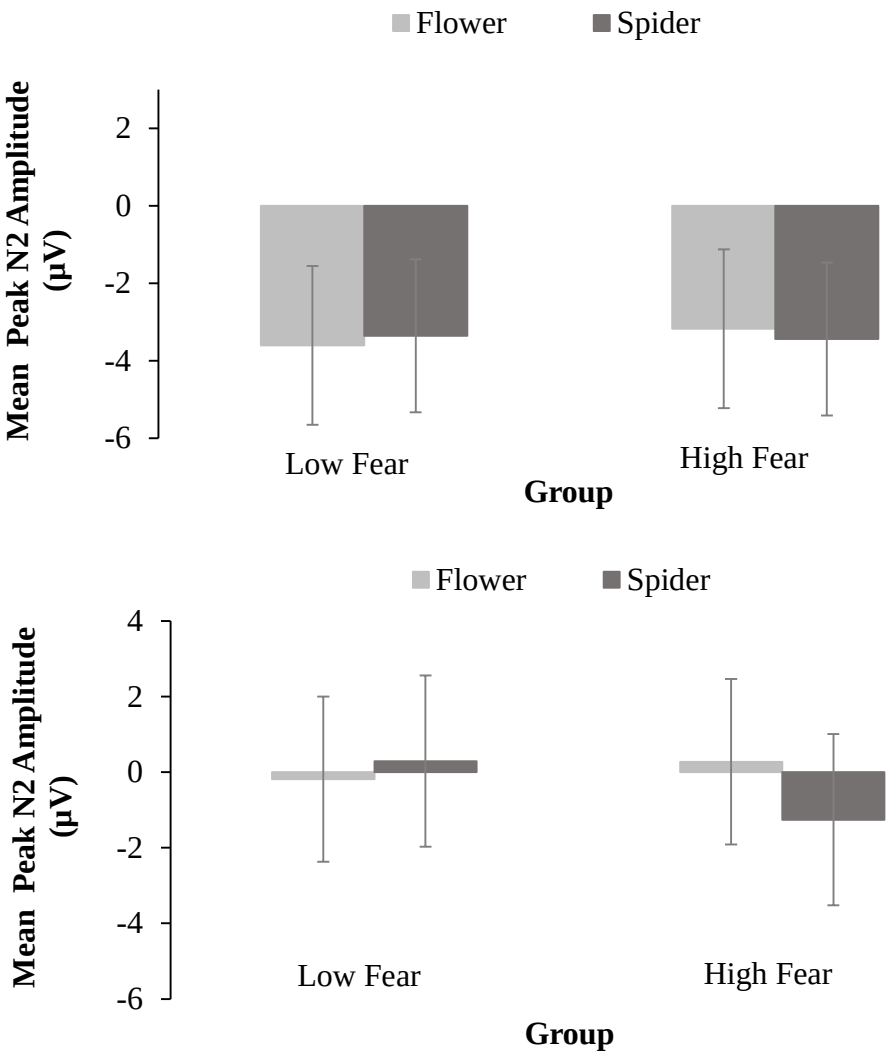

Figure 10. Mean peak N2 amplitude for low and high fear participants at Fz for flower and spider flankers in incongruent image nogo (top) and go (bottom) conditions (error bars represent 95% CIs).

SUDS

A 2 (Group: high fear, low fear) x 4 (SUDS rating block: one, two, three, four) mixed ANOVA was conducted on participants' SUDS ratings. Cell means are shown in Table 6. The main effect of Group was non-significant, $F(1,28)=1.21$, $p=.281$, $\eta_p^2=.041$, as was the main effect of SUDS rating block, $F(2, 47)=2.93$, $p=.072$, $\eta_p^2=.095$, Greenhouse-Geisser corrected, and the Group x SUDS rating block interaction, $F(2, 47)=2.45$, $p=.106$, $\eta_p^2=.080$, Greenhouse-Geisser corrected.

Table 6

Mean SUDs Ratings for each Task Block with SD in Parentheses and 95% Confidence Intervals for Low and High Fear Groups

	Low Fear		High Fear	
SUDs Block	*M* (*SD*)	95% CI	*M* (*SD*)	95% CI
One	4.0 (7.1)	[0.3, 7.7]	3.7 (6.9)	[-0.1, 7.4]
Two	5.7 (8.2)	[0.8, 10.6]	7.7 (10.2)	[2.8, 12.6]
Three	4.7 (5.2)	[-1.7, 11.1]	10.3 (16.3)	[3.9, 16.7]
Four	4.3 (6.2)	[-1.5, 10.1]	11.0 (14.2)	[5.2, 16.8]

Discussion

The aim of the present study was to further investigate the role of hypervigilance and inhibitory control in specific fear using a task with greater cognitive load. The hypotheses that high relative to low spider fear participants would demonstrate specific hypervigilance through faster RTs and greater P1 amplitude in response to spider relative to flower targets on go trials were not supported. Behaviourally, all participants, regardless of fear status and flanker type, were faster to respond to spider targets than flower targets. Electrophysiologically, the absence of a significant Group x Target interaction indicated neither high or low fear groups showed significant differences in P1 amplitude in response to spider and flower targets, with the only between-group difference being significantly less P1 amplitude in the left relative to right hemisphere for low relative to high fear participants.

The hypothesis that high relative to low fear participants would demonstrate behavioural interference through slower RTs in response to flower targets flanked by spider distractors as compared to flower targets flanked by neutral dashes was not supported. While the Flanker type x Group x Target interaction was non-significant, overall, RTs were slower in response to incongruent relative to neutral flanker trials for both groups, regardless of whether the trial was spider or flower incongruent. The hypothesis that high relative to low fear participants would have reduced N2 amplitude for spider relative to flower flankers in nogo relative to go trials, as evidence of reduced inhibitory control, was also not supported. However, there was an unanticipated increase in N2 amplitude in response to trials with spider flankers for the high but not the low fear group that trended towards significance. This effect did not differ for nogo or go trials overall.

There was no evidence for a speed-accuracy trade-off in the behavioural data, as there were no Target image effects on accuracy for the go analysis. Both lower accuracy and longer RTs were observed for incongruent trials, suggesting unanimous decline in both measures of performance.

Hypervigilance to Feared Stimuli

The finding of faster RTs to spider targets in both groups diverges from previous research. Facilitated RTs to spider relative to neutral images specifically in individuals with a high fear/phobia of spiders relative to controls has been shown in picture identification (Kolassa, Musial, Mohr, Trippe, & Milner, 2005) and dot probe tasks (Lipp & Derakshan, 2005), and a previous flanker go nogo task (Venettacci, Johnstone, Kirkby, & Matthews, 2016). These findings have been interpreted in accordance to theory proposed by Eysenck (1992) which suggests this process represents a rapid narrowing of attention to threatening stimuli in high anxiety individuals (Kolassa et al., 2005; Venettacci et al., 2016). The current finding may indicate an evolutionary attentional mechanism in response to potentially dangerous stimuli was evoked in both high and low fear individuals rather than the expected fear-specific response in the high fear group. Ohman and Mineka (2001) proposed a neural-based fear module organised around the amygdala that is automatically activated in response to evolutionarily-relevant fear stimuli associated with threat. Evidence for this has been shown by healthy participants exhibiting faster responses to evolutionary-relevant threats (i.e., snakes, spiders) compared to fear-irrelevant objects (Blanchette, 2006).

The finding of no differences between groups for P1 amplitude across target images also does not provide support for specific hypervigilance. This is in contrast to a recent finding of increased P1 amplitude for high relative to low fear

participants specifically in response to spider relative to flower targets (Venettacci, Johnstone, Kirkby, & Matthews, 2016). The present finding is also not in line with previous evidence of general hypervigilance where spider phobia participants relative to controls have shown greater P1 amplitude for both neutral and spider images (e.g., Kolassa et al., 2007), and does not reconcile with behavioural evidence of a potential evolutionary attentional mechanism. Thus, P1 data does not support a cortical mechanism related to early automatic capture of attention in fearful individuals or towards feared stimuli (Hofmann, Ellard, & Siegle, 2012) supported by the bottom-up component of the orienting attentional network (Petersen & Posner, 2012).

The P1 finding of reduced amplitude in the left occipital site in low relative to high fear participants was unanticipated. It is possible that this reflects group differences in processing of the task stimuli. Evidence of hemispheric asymmetry in visual processing has indicated global processing of visual information as a whole and local processing for the independent features for a visual scene is dominant for the right and left visual cortex, respectively (Christie et al., 2012; Hellige, 1996). Both local and global visual processing has been found to modulate the P1 component (Batty & Taylor, 2003; Han, He, Yund, & Woods, 2001). Studies have found an association between trait anxiety and a bias towards using local visual processing (Basso, Schefft, Ris, & Dember, 1996; Derryberry & Reed, 1998). In the current study, high but not low fear participants were overall less accurate to go and nogo trials where incongruent flankers featured and showed a difference in N2 amplitude for spider relative to flower flankers. Accordingly, the reduction in P1 amplitude for the left hemisphere in low fear participants may reflect differential engagement in local processing of distractors throughout the task that varied

according to fear status.

Taken together, behavioural and electrophysiological findings for the hypervigilance hypotheses do not provide support for predictions made by ACT in specific fear. According to ACT, anxiety stimulates a stimulus-driven attentional network that resembles the bottom-up component of the orienting network proposed by Petersen and Posner (2012), serving to facilitate attentional bias to threat (Eysenck, Derakshan, Santos, & Calvo, 2007). Venettacci, Johnstone, Kirkby, and Matthews (2016) were the first to find behavioural and electrophysiological evidence of this in the form of specific hypervigilance in specific fear, which suggested attentional bias to feared stimuli in specific fear/phobia is underpinned by early attentional selections. The finding provided original empirical support for a possible role of this mechanism in the etiology and maintenance of specific fear/phobia, which could potentially be targeted in treatment interventions. However, using a modified flanker go nogo task designed to increase cognitive load, the finding was not replicated. This may indicate that this it is not robust and that specific hypervigilance may not be a critical characteristic of specific fear/phobia.

An alternative explanation for the absence of specific hypervigilance in high fear participants may be that cognitive load associated with the modified task induced interference effects that masked facilitation effects. Facilitation effects have typically been observed in paradigms with low cognitive load such as picture identification (Kolassa, Musial, Mohr, Trippe, & Milner, 2005), and dot probe tasks (Mogg & Bradley, 2006). A recent study used an Attention Network Test where preceding invalid visual spatial cues are argued to increase cognitive load in a flanker task (Flynn, 2015). High relative to low spider fear participants did not show hypervigilance through facilitated RTs or enhanced P1 amplitude following valid

spider cues. However, interference effects were found for high fear participants on incongruent flanker trials proceeded by spider cues. In the present study, it is possible that increased demands on the executive control network and use of incongruent trials only (i.e., the need to ignore incompatible flankers on every trial), required disproportionate engagement of voluntary attentional processing which limited the ability to capture differences in stimulus driven processing between the groups. This may help to explain the reduced accuracy on incongruent go and nogo trials (indicative of interference) coupled with a lack of evidence of hypervigilance in the high fear group. Possibly, the contrast between neutral and incongruent flankers may have enhanced the difficulty of the latter, as the overall reduction in accuracy for these trials may indicate. Future research could explore this by removing neutral dash flanker trials and re-including congruent trials in a similar paradigm with more objects to increase cognitive load to further examine whether evidence of both increased stimulus-driven and decreased goal-directed attentional processing can be found in specific fear.

It is also possible that the lack of hypervigilance was due to the current paradigm not adequately eliciting fear responses in high fear participants. While the groups differed significantly on measures of spider fear, with very large effect sizes noted, the groups did not differ in state anxiety (measured via SUDS) during the task at any of the four blocks. Additionally, follow-up bivariate correlation analyses (not reported) revealed no significant relationship between SPQ scores and RT or P1 amplitude for spider targets among high or low fear participants. It is possible that the increased cognitive load in the current paradigm contributed to this. For example, Vytal, Cornwell, Arkin, and Grillon (2012) found anxiety was reduced during a high relative to low load task. Future research could aim to overcome this

by using real spider images, which would more closely resemble the feared stimulus and likely elicit greater fear. Additionally, the sample used in the current study was not explicitly clinically defined. Thus, the overall level of intensity of fear symptoms may not have been sufficient to yield hypervigilance effects in the current task. Thus, future research should aim to use a clinical sample.

Interference and Inhibitory Control

Fear-related behavioural interference in high fear participants was not observed. In emotional Stroop tasks, slower colour-naming latencies have been found in response to spider-related words in paradigms using five colours (e.g., Kwakkenbos, Becker, & Rinck, 2010), but not in response to real images of spiders where only two colours were used (Kolassa, Musial, Kolassa, & Miltner, 2006; Kolassa, Musial, Mohr, Trippe, & Milner, 2005), possibly due to differences in cognitive load. Additionally, fear-related interference has not been observed in flanker tasks with relatively low levels of cognitive load (e.g., Venettacci, Johnstone, Kirkby, & Matthews, 2016). Given that ACT predicts anxiety is more likely to impair inhibitory control in the presence of threat distractors when task difficulty is greater (Eysenck, Derakshan, Santos, & Calvo, 2007), it was expected that high fear participants would exhibit fear-specific behavioural interference in the current paradigm where cognitive load had been targeted. However, this was not found.

However, there was an overall decline in accuracy and increase in RT and P1 amplitude for go trials with incongruent relative to neutral flankers, irrespective of image. Thus, this more complex type of flanker may have received greater visual attention at an early stage of processing, resulting in greater interference in performance. This may reflect activation of the dorsal attentional system of the

orienting network, which can facilitate rapid top-down control of attention to prepare for expected input (Petersen & Posner, 2012). Further, the finding may be suggestive of a general, non-fear related interference in both groups. However, high fear participants showed an overall reduction in accuracy for go and nogo trials with incongruent flankers, suggesting this flanker type greater affected their performance compared to low fear participants. Differences in accuracy were not hypothesised as ACT suggests that anxiety should greater impair task efficiency, with a decrement in task effectiveness prevented through increased effort or compensatory strategies (Eysenck, Derakshan, Santos, & Calvo, 2007). The current findings therefore provides some support for a reduction in goal-directed attentional processing associated with disrupted inhibitory control in high fear individuals, albeit not in response to the fear-relevant distractors as expected.

A possible explanation for an absence of fear-specific behavioural interference in high fear participants may relate to the finding that high but not low fear participants showed increased N2 amplitude on incongruent trials with spider compared to flower flankers (for both go and nogo trials). This finding extends from a previous study using a flanker go nogo task where no N2 effects were observed in high spider fear participants (Venettacci, Johnstone, Kirkby, & Matthews, 2016), possibly due to a lack of cognitive load. However, the finding diverges from evidence of reduced nogo-N2 in clinical groups where impaired inhibitory control is thought to be characteristic (Kim, Kim, Yoo, & Kwon, 2007; Thomas, Gonsalvez, & Johnstone, 2014). While both groups in the current study had reduced accuracy on incongruent go and nogo trials for spider relative to flower flankers, greater N2 amplitude on these trials for high fear participants may indicate they used more inhibitory resources to perform behaviourally at the same level as low fear

participants for these conditions. This may indicate use of a compensatory inhibitory process employed through the executive control network in high fear individuals which could have prevented fear-specific behavioural interference in the RT analysis.

While overall nogo trials had greater N2 amplitude, the difference in N2 for spider and flower flanker trials did not vary as a function of trial type. Therefore, the current findings do not represent nogo-N2 as predicted. It is possible that the finding instead represents an interference suppression mechanism, whereby high fear participants exerted more cognitive effort to resist interference from the spider distractors (Brydges et al., 2012). Interference suppression can modulate N2 amplitude at the midline frontal site Fz, but with less amplitude, greater latencies, and more central distribution than nogo-N2 (Brydges et al., 2012). This mechanism likely involves resolving preparation of an incorrect response that arises from conflict induced by incongruent flankers (Folstein & Van Petten, 2008). In the current study, the overall decline in accuracy for trials with spider flankers may suggest reduced ability to resolve conflict as both groups demonstrated a facilitated response to these stimuli when presented as targets on go trials. The N2 finding may then indicate greater use of inhibitory resources to resolve conflict for feared relative to neutral distractors among high fear participants.

Together, behavioural and electrophysiological findings provide some evidence for predictions made by ACT. Less accuracy for image incongruent trials in high fear participants may indicate a reduction in goal-directed attentional processing (Eysenck, Derakshan, Santos, & Calvo, 2007). However this was not specifically in response to spider distractors and is therefore not suggestive of a process relating to attentional bias to feared stimuli. The possibility that high fear

participants exerted more cognitive effort to suppress interference from spiders could be interpreted as evidence for ACT's prediction that anxiety reduces the efficiency of inhibitory control when threat-distractors are present (Eysenck et al., 2007). However, this is unexpected given the cognitive load added to this task, as ACT proposes it becomes harder for anxious individuals to use greater resources to sustain performance when task demands are greater (Eysenck et al., 2007). The finding is also not concurrent with under-recruitment of frontal regions in regulating attentional biases proposed by Bishop (2007). However, the findings may relate to failure to adequately elicit fear-related arousal in the high fear group, as discussed earlier.

The N2 finding should be interpreted with caution. The overall N2 interaction only trended towards significance and the effect size for the high fear group was small in magnitude. Thus, future research should endeavour to replicate this finding before strong conclusions are drawn. If robust, the finding could inform directions for clinical treatment of specific fear/phobia. Attentional biases are one mechanism thought to be involved in impaired inhibitory learning (needed for successful extinction of fear) in individuals with an anxiety disorder due to inhibitory deficits (Craske, 2015; Craske, Liao, Brown, & Vervliet, 2012). Following attentional training away from threat, anxious individuals have shown increased N2 amplitude, suggesting improved inhibitory control (Eldar & Bar-Haim, 2010). Accordingly, N2 amplitude may have utility as an electrophysiological means to quantifying increased efficiency of inhibitory control as attentional biases attenuate in specific fear. However, future research should first determine whether reduced inhibitory control is observed when attentional biases are present.

A potential threat to internal validity is the significantly higher psychological

distress scores in the high fear group. However, a preliminary ANCOVA was run with psychological distress entered as a covariate and revealed that this variable did not significantly relate to any of the dependent variables in any analysis. Additionally, it is argued that this analysis would not be appropriate given participants were not randomly allocated to groups, making it difficult to establish whether group differences at pre-test were attributable to random error or true group differences (Miller & Chapman, 2001). Given psychological distress is typical in anxiety-related disorders, it is unlikely to be partialled out independently of the effect of spider fear (Miller & Chapman, 2001). Future research with group differences in psychological characteristics could establish a clearer baseline of performance by including a behavioural pre-task free from spider stimuli.

Summary and Conclusions

The present study investigated behavioural and electrophysiological correlates of hypervigilance and inhibitory control in specific fear using a flanker go nogo task which was modified to increase cognitive load. Both high and low fear groups demonstrated behavioural specific hypervigilance indexed by faster RTs to spider targets, suggestive of an evolutionary attentional mechanism. Specific hypervigilance was not evident in P1 data, with only a reduction in P1 amplitude observed in the left hemisphere for low fear participants, which may be indicative of less reliance on local visual processing compared to the high fear group. Fear-specific behavioural interference did not surface specifically in high fear participants. While they exhibited less accuracy overall on incongruent image trials, a reduction in accuracy for trials with spider flankers was observed in both groups. Increased N2 amplitude on spider flanker trials for high fear participants may indicate the use of a compensatory inhibitory process to suppress interference from

irrelevant fear-related information.

While the current investigation sought to further examine the unique and interactive roles of hypervigilance and inhibitory control as potential mechanisms underlying attentional biases to feared stimuli in individuals with specific fear, no such biases were observed. Inconsistent with ACT (Eysenck, Derakshan, Santos, & Calvo, 2007), there was an absence of increased automatic attentional processing to feared stimuli that was unique to high fear participants. Explanations for this include the possibility that fear was not sufficiently elicited in high fear participants and that the increased cognitive load with the current task disproportionally engaged voluntary processing, masking hypervigilance effects. Subsequently, the lack of hypervigilance might explain why high fear participants also did not show greater interference compared to low fear participants, or reduced inhibitory control, in response to spider distractors. Possibly, while requiring more cognitive effort, high fear participants were able to recruit the sufficient resources to prevent greater decrement to goal-directed attentional processing compared to low fear participants, when exposed to their feared object. This is a relatively novel finding of enhanced performance of the executive control attentional network (Petersen & Posner, 2012) in specific fear and while in line with ACT's prediction that anxiety impairs the efficiency of inhibitory control, it does not extend the assumptions of ACT to show that this manifests in the form of an attentional bias towards feared stimuli in specific fear (Eysenck et al., 2007). However, as discussed, this finding needs replication. Additionally, the exact role of fear and the interaction between the orienting and executive control networks cannot be inferred from the current study. Future research should aim to further investigate this using paradigms better placed to elicit fear responses and engage both automatic and voluntary attentional

processing. This would help to ascertain whether increased automatic and decreased voluntary attentional processing takes place concurrently in specific fear, or whether they are observable only in isolation.

References

Åkerstedt, T., & Gillberg, M. (1990). Subjective and objective sleepiness in the active individual. *International Journal of Neuroscience, 52,* 29-37. doi: 10.3109/00207459008994241

Anderson, A. K., Christoff, K., Panitz, D., De Rosa, E., & Gabrieli, J. D. E. (2003). Neural correlates of the automatic processing of threat facial signals. *The Journal of Neuroscience, 23*, 5627–5633.

Antony, M. M., Bieling, P. J., Cox, B. J., Enns, M. W., & Swinson, R. P. (1998). Psychometric properties of the 42-Item and 21-item versions of the Depression Anxiety Stress Scales in clinical groups and a community sample. *Psychological Assessment, 10*, 176-181. doi: 10.1037/1040-3590.10.2.176

Babor, T. F., Higgins-Biddle, J. C., Saunders, J. B., & Monteiro, M. G. (2001). *The Alcohol Use Disorders Identification Test: Guidelines for use in primary care* (2nd ed.). Geneva: World Health Organization.

Bar, M. (2003). A cortical mechanism for triggering top-down facilitation in visual object recognition. *Journal of Cognitive Neuroscience, 15*, 600–609. doi:10.1162/089892903321662976

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: A meta-analytic study. *Psychological Bulletin, 133*, 1-24. doi:10.1037/0033- 2909.133.1.1

Basso, M. R., Schefft, B. K., Ris, M. D., & Dember, W. N. (1996). Mood and global-local visual processing. *Journal of the International Neuropsychological Society, 2,* 249- 255. doi: 10.1017/S1355617700001193

Batty, M., & Taylor, M. J. (2003). Early processing of the six basic facial emotional expressions. *Cognitive Brain Research, 17*, 613-620. doi: 10.1016 /S0926-6410(03)00174-5

Bishop, S. J. (2007). Neurocognitive mechanisms of anxiety: An integrative account. *Trends in Cognitive Sciences, 11*, 307-316. doi:10.1016/j.tics.2007.05.008

Bishop, S. J., Duncan, J., Brett, M., & Lawrence, A. D. (2004). Prefrontal cortical function and anxiety: Controlling attention to threat-related stimuli. *Nature Neuroscience, 7*, 184-188. doi:10.1038/nn1173

Blanchette, I. (2006). Snakes, spiders, guns, and syringes: How specific are evolutionary constraints on the detection of threatening stimuli? *The Quarterly Journal of Experimental Psychology*, 59, 1484-504. doi: 10.1080/02724980543000204

Bohn, M. J., Babor, T. F., & Kranzler, H. R. (1995). The Alcohol Use Disorders Identification Test (AUDIT): Validation of a screening instrument for use in medical settings. *Journal of Studies on Alcohol, 56*, 423-432. doi: 10.15288/jsa.1995.56.423

Boschen, M. J., Neumann, D. L., & Waters, A. M. (2009). Relapse of successfully treated anxiety and fear: Theoretical issues and recommendations for clinical practice. *Australian and New Zealand Journal of Psychiatry, 43*, 89-100. doi:10.1080/00048670802607154 23

Bruin, K. J., & Wijers, A. A. (2002). Inhibition, response mode, and stimulus probability: A comparative event-related potential study. *Clinical Neurophysiology, 113*, 1172-1182. doi: 10.1016/S1388-2457(02)00141-4

Brydges, C. R., Clunies-Ross, K., Clohessy, M., Lo, Z. L., Nguyen, A., Rousset, C., . . . Fox, A. M. (2012). Dissociable components of cognitive control: An event-related potential (ERP) study of response inhibition and interference suppression. *PLoS One, 7*, e34482. doi:10.1371/journal.pone.0034482

Callejas, A., Lupianez, J., Funes, M. J., & Tudela, P. (2005). Modulations among the alerting, orienting and executive control networks. *Experimental Brain Research, 167*, 27-37. doi:10.1007/s00221-005-2365-z

Carlsson, K., Petersson, K. M., Lundqvist, D., Karlsson, A., Ingvar, M., & Ohman, A. (2004). Fear and the amygdala: Manipulation of awareness generates differential cerebral responses to phobic and fear-relevant (but nonfeared) stimuli. *Emotion, 4*, 340-353. doi:10.1037/1528-3542.4.4.340

Christie, J., Ginsberg, J. P., Steedman, J., Fridriksson, J., Bonilha, L., & Rorden, C. (2012). Global versus local processing: Seeing the left side of the forest and the right side of the trees. *Frontiers in Human Neuroscience, 6*, 1-8. doi: 10.3389/fnhum.2012.00028

Cisler, J. M., & Koster, E. H. W. (2010). Mechanisms of attentional biases towards threat in anxiety disorders: An integrative review. *Clinical Psychology Review, 30*, 203-216. doi:10.1016/j.cpr.2009.11.003

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). New York: Routledge.

Cohen, J. (1992). A power primer. *Psychological Bulletin, 112*, 155-159. doi: 10.1037/0033- 2909.112.1.155

Craske, M. G. (2015). Optimizing exposure therapy for anxiety disorders: An inhibitory learning and inhibitory regulation approach. *Verhaltenstherapie, 25*, 134-143. doi:10.1159/000381574

Craske, M. G., Liao, B., Brown, L., & Vervliet, B. (2012). Role of inhibition in exposure therapy. *Journal of Experimental Psychopathology, 3*, 322-345. doi:10.5127/jep.026511

Creamer, M., Foran, J., & Bell, R. (1995). The Beck Anxiety Inventory in a non-clinical sample. *Behaviour Research and Therapy, 33*, 477-485. doi: 10.1016/0005- 7967(94)00082-U

Derryberry, D., & Reed, A. (1998). Anxiety and attentional focusing: Trait, state and hemispheric influences. *Personality and Individual Differences, 25*, 745-761. doi: 10.1016/S0191-8869(98)00117-2

Derryberry, D., & Reed, A. (2002). Anxiety-related attentional biases and their regulation by attentional control. *Journal of Abnormal Psychology, 111*, 225-236. doi:10.1037/0021-843x.111.2.225

Di Russo, F., Martinez, A., Sereno, M. I., Pitzalis, S., & Hillyard, S. A. (2002). Cortical sources of the early components of the visual evoked potential. *Human Brain Mapping, 15*, 95-111. doi:10.1002/hbm.10010

Dye, M. W. G., Green, C. S., & Bavelier, D. (2009). The development of attention skills in action video game players. *Neuropsychologia, 47*, 1780-1789. doi: 10.1016/j.neuropsychologia.2009.02.002

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon identification of a target letter in a non search task. *Perception and Psychophysics*, *16*, 143-149. doi:10.3758/BF03203267

Eysenck, M. W. (1992). *Anxiety: The cognitive perspective*. East Sussex: Lawrence Erlbaum Associates.

Eysenck, M. W., Derakshan, N., Santos, R., & Calvo, M. G. (2007). Anxiety and cognitive performance: Attentional control theory. *Emotion, 7*, 336-353. doi:10.1037/1528-3542.7.2.336

Falkenstein, M. (2006). Inhibition, conflict and the nogo-N2. *Clinical Neurophysiology, 117*, 1638-1640. doi:10.1016/j.clinph.2006.05.002

Falkenstein, M., Hoormann, J., & Hohnsbein, J. (1999). ERP components in Go/Nogo tasks and their relation to inhibition. *Acta Psychologica, 101*, 267-291. doi:10.1016/S0001-6918(99)00008-6

Fan, J., Gu, X., Guise, K. G., Liu, X., Fossella, J., Wang, H., & Posner, M. I. (2009). Testing the behavioral interaction and integration of attentional networks. *Brain and Cognition, 70*, 209-220. doi:10.1016/j.bandc.2009.02.002

Fenske, M. J., & Eastwood, J. D. (2003). Modulation of focused attention by faces expressing emotion: Evidence from flanker tasks. *Emotion, 3*, 327-343. doi:10.1037/1528-3542.3.4.327

Finucane, A. M. (2011). The effect of fear and anger on selective attention. *Emotion, 11*, 970-974. doi:10.1037/a0022574

Flynn, S. (2015). *Behavioural and neutral correlates of orienting and executive control in high and low spider fear groups* (Unpublished thesis). University of Tasmania, Hobart.

Folstein, J. R., & Van Petten, C. (2008). Influence of cognitive control and mismatch on the N2 component of the ERP: A review. *Psychophysiology, 45*, 152-170. doi:10.1111/j.1469-8986.2007.00602.x

Gerdes, A. B., Alpers, G. W., & Pauli, P. (2008). When spiders appear suddenly: Spider-phobic patients are distracted by task-irrelevant spiders. *Behaviour Research and Therapy, 46*, 174-187. doi:10.1016/j.brat.2007.10.010

Han, S., He, X., Yund, W., & Woods, D. L. (2001). Attentional selection in the processing of hierarchical patterns: An ERP study. *Biological Psychology, 56*, 113-130. doi: 10.1016/S0301-0511(01)00073-4

Heil, M., Osman, A., Wiegelmann, J., Rolke, B., & Hennighausen, E. (2000). N200 in the Eriksen-task: Inhibitory executive processes? *Journal of Psychophysiology, 14*, 218-225. doi:10.1027//0269-8803.14.4.218

Hellige, J. B. (1996). Hemispheric asymmetry for visual information processing. *Acta Neurobiologiae Experimentalis, 56*, 485-497.

Hofmann, S. G., Ellard, K. K., & Siegle, G. J. (2012). Neurobiological correlates of cognitions in fear and anxiety: A cognitive-neurobiological information-processing model. *Cognition and Emotion, 26*, 282-299. doi:10.1080/02699931.2011.579414

Howell, D. C. (2012). *Statistical methods for psychology* (8th ed.). Belmont, California: Cengage Learning.

Janak, P. H., & Tye, K. M. (2015). From circuits to behaviour in the amygdala. *Nature, 517*, 284-292. doi:10.1038/nature14188

Jodo, E., & Kayama, Y. (1992). Relation of a negative ERP component to response inhibition in a go/no-go task. *Electroencephalography and Clinical Neurophysiology, 82*, 477-482. doi:10.1016/0013-4694(92)90122-X

Kanske, P., & Kotz, S. A. (2010). Modulation of early conflict processing: N200 responses to emotional words in a flanker task. *Neuropsychologia, 48*, 3661-3664. doi:10.1016/j.neuropsychologia.2010.07.021

Kessler, R. C., Andrews, G., Colpe, L. J., Hiripi, E., Mroczek, D. K., Normand, S. L. T., . . . Zaslavsky, A. M. (2002). Short screening scales to monitor

population prevalences and trends in non-specific psychological distress. *Psychological Medicine, 32,* 959-976. doi: 10.1017}S0033291702006074

Kim, M. S., Kim, Y. Y., Yoo, S. Y., & Kwon, J. S. (2007). Electrophysiological correlates of behavioral response inhibition in patients with obsessive-compulsive disorder. *Depression and Anxiety, 24*, 22-31. doi:10.1002/da.20195

Kolassa, I., Buchmann, A., Lauche, R., Kolassa, S., Partchev, I., Miltner, W. H. R., & Musial, F. (2007). Spider phobics more easily see a spider in morphed schematic pictures. *Behavioral and Brain Functions, 3*, 59. doi:10.1186/1744-9081-3-59

Kolassa, I., Musial, F., Kolassa, S., & Miltner, W. H. R. (2006). Event-related potentials when identifying or color-naming threatening schematic stimuli in spider phobic and non-phobic individuals. *BMC Psychiatry, 6*, 38. doi:10.1186/1471-244X-6-38

Kolassa, I., Musial, F., Mohr, A., Trippe, R. H., & Miltner, W. H. R. (2005). Electrophysiological correlates of threat processing in spider phobics. *Psychophysiology, 42*, 520-530. doi:10.1111/j.1469-8986.2005.00315.x

Kwakkenbos, L., Becker, E. S., & Rinck, M. (2010). Fear of spiders: The effect of real threat on the interference caused by symbolic threat. *Cognition and Emotion, 24*, 800-809. doi:10.1080/02699930902995901

Lavie, N., Hirst, A., de Fockert, J. W., & Viding, E. (2004). Load theory of selective attention and cognitive control. *Journal of Experimental Psychology: General, 133*, 339-354. doi:10.1037/0096-3445.133.3.339

Laviers, E. G., Burton, M. P., & McKeefry, D. J. (2007). The cortical topography of visual evoked potentials elicited by chromatic and luminance motion. *The*

Open Ophthalmology Journal, 1, 25-34. doi:10.2174/1874364100701010025

Lavy, E., van den Hout, M., & Arntz, A. (1993). Attentional bias and facilitated escape: A pictorial test. *Advances in Behaviour Research and Therapy, 15*, 279-289. doi:10.1016/0146-6402(93)90013-R

Li, X., Li, X., & Luo, Y. (2005). Anxiety and attentional bias for threat: An event-related potential study. *NeuroReport, 16*, 1501-1505. doi:10.1097/01.wnr.0000176522.2
6971.83

Lipp, O. V., & Derakshan, N. (2005). Attentional bias to pictures of fear-relevant animals in a dot probe task. *Emotion, 5*, 365-369. doi:10.1037/1528-3542.5.3.365

Lipp, O. V., & Waters, A. M. (2007). When danger lurks in the background: Attentional capture by animal fear-relevant distractors is specific and selectively enhanced by animal fear. *Emotion, 7*, 192-200. doi:10.1037/1528-3542.7.1.192

Luck, J. S. (2014). *An introduction to the event-related potential technique*. London, England: The MIT Press.

Mangun, G. R. (1995). Neural mechanisms of visual selective attention. *Psychophysiology, 32*, 4-18. doi:10.1111/j.1469-8986.1995.tb03400.x

Mathews, A., & MacLeod, C. (2002). Induced processing biases have causal effects on anxiety. *Cognition and Emotion, 16*, 331-354. doi:10.1080/02699930143000518

Michalowski, J. M., Melzig, C. A., Weike, A. I., Stockburger, J., Schupp, H. T., & Hamm, A. O. (2009). Brain dynamics in spider-phobic individuals exposed to phobia-relevant and other emotional stimuli. *Emotion, 9*, 306-315.

doi:10.1037/a0015550

Miller, G. A., & Chapman, J. P. (2001). Misunderstanding analysis of covariance. *Journal of Abnormal Psychology, 110*, 40-48. doi:10.1037/0021-843X.110.1.40

Miltner, W. H., R., Trippe, R. H., Krieschel, S., Gutberlet, I., Hecht, H., & Weiss, T. (2005). Event-related brain potentials and affective responses to threat in spider/snake-phobic and non-phobic subjects. *International Journal of Psychophysiology, 57*, 43-52. doi: 10.1016/j.ijpsycho.2005.01.012

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex "Frontal Lobe" tasks: A latent variable analysis. *Cognitive Psychology, 41*, 49-100. doi:10.1006/cogp.1999.0734

Mogg, K., & Bradley, B. P. (2006). Time course of attentional bias for fear-relevant pictures in spider fearful individuals. *Behaviour Research and Therapy, 44*, 1241-1250. doi:10.1016/j.brat.2006.05.003

Morris, J. S., Friston, K. J., Büchel, C., Frith, C. D., Young, A. W., Calder, A. J., & Dolan, R. J. (1998). A neuromodulatory role for the human amygdala in processing emotional facial expressions. *Brain, 121*, 47–57. doi:10.1093/brain/121.1.47

Muris, P., & Merckelbach, H. (1996). A comparison of two spider fear questionnaires. *Journal of Behavioral Therapy and Experimental Psychiatry, 27*, 241-244. doi:10.1016/S0005-7916(96)00022-5

Nee, D. E., Wager, T. D., & Jonides, J. (2007). Interference resolution: Insights from a meta-analysis of neuroimaging tasks. *Cognitive, Affective, and Behavioral Neuroscience, 7*, 1-17. doi:10.3758/CABN.7.1.1

Nieuwenhuis, S., Yueng, N., van den Wildenberg, W., & Ridderinkhof, K. R. (2003). Electrophyiological correlates of anterior cingulate function in a go/no-go task: Effects of response conflict and trial type frequency. *Cognitive, Affective and Behavioral Neuroscience, 3*, 17-26. doi:10.3758/CABN.3.1.17

Öhman, A. (2008). Fear and anxiety: Overlaps and dissociations In M. Lewis, J. M. Haviland-Jones, & L. F. Barrett (Eds.), *Handbook of emotions* (3rd ed., pp. 709-729). New York: The Guilford Press.

Öhman, A., & Mineka, S. (2001). Fears, phobias, and preparedness: Toward an evolved module of fear and fear learning. *Psychological Review, 108*, 483-522. doi: 10.1037//0033-295X.108.3.483

Oosterink, F. M., de Jongh, A., & Hoogstraten, J. (2009). Prevalence of dental fear and phobia relative to other fear and phobia subtypes. *European Journal of Oral Sciences, 17*, 135-143. doi:10.1111/j.1600-0722.2008.00602.x

O'Toole, L., & Dennis, T. A. (2012). Attention training and the threat bias: An ERP study. *Brain and Cognition, 78*, 63-73. doi:10.1016/j.bandc.2011.10.007

Petersen, S. E., & Posner, M. I. (2012). The attention system of the human brain: 20 years after. *Annual Review of Neuroscience, 35*, 73-89. doi:10.1146/annurev-neuro-062111- 150525

Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience, 13,* 25-42.doi:10.1146/annurev.ne. 13.030190.000325

Railo, H., Salminen-Vaparanta, N., Henriksson, L., Revonsuo, A., & Koivisto, M. (2012). Unconscious and conscious processing of color rely on activity in

early visual cortex: A TMS study. *Journal of Cognitive Neuroscience, 24*, 819–829. doi:10.1162/jocn_a_00172

Rinck, M., Reinecke, A., Ellwart, T., Heuer, K., & Becker, E. S. (2005). Speeded detection and increased distraction in fear of spiders: Evidence from eye movements. *Journal of Abnormal Psychology*, *114*, 235-248. doi:10.1037/0021-843X.114.2.235

Schmajuk, M., Liotti, M., Busse, L., & Woldorff, M. G. (2006). Electrophysiological activity underlying inhibitory control processes in normal adults. *Neuropsychologia, 44*, 384-395. doi:10.1016/j.neuropsychologia.2005.06.005

Spielberger, C. D. (1983). *State-Trait Anxiety Inventory manual*. Redwood City, CA: Mind Garden Inc.

Szymanski, J., & O'Donohue, W. (1995). Fear of Spiders Questionnaire. *Journal of Behavior Therapy and Experimental Psychiatry, 26*, 31-34. doi:10.1016/0005-7916(94)00072-T

Thomas, S. J., Gonsalvez, C. J., & Johnstone, S. J. (2014). How specific are inhibitory deficits to obsessive-compulsive disorder? A neurophysiological comparison with panic disorder. *Clinical Neurophysiology, 125*, 463-475. doi:10.1016/j.clinph.2013.08.018

Thorpe, S. J., & Salkovskis, P. M. (1997). Information processing in spider phobics: The Stroop colour naming task may indicate strategic but not automatic attentional bias. *Behavior Research and Therapy, 35*, 131-144. doi:10.1016/S0005-7967(96)00093-9

Van Bockstaele, B., Verschuere, B., Tibboel, H., De Houwer, J., Crombez, G., & Koster, E. H. (2014). A review of current evidence for the causal impact of

attentional bias on fear and anxiety. *Psychological Bulletin, 140,* 682-721. doi:10.1037/a0034834

Venettacci, R., Johnstone, A., Kirkby, K. C., & Matthews, A. (2016). *ERP correlates of attentional processing in spider fear: Evidence of threat-specific hypervigilance*. Manuscript submitted for publication.

Vuilleumier, P., Armony, J. L., Driver, J., & Dolan, R. J. (2001). Effects of attention and emotion on face processing in the human brain: An event-related fMRI study. *Neuron, 30,* 829-841. doi:10.1016/S0896-6273(01)00328-2

Vytal, K., Cornwell, B., Arkin, N., & Grillon, C. (2012). Describing the interplay between anxiety and cognition: From impaired performance under low cognitive load to reduced anxiety under high load. *Psychophysiology, 49,* 842-852. doi: 10.1111/j.1469-8986.2012.01358.x

Watts, F. N., McKenna, F. P., Sharrock, R., & Trezise, L. (1986). Colour naming of phobia-related words. *British Journal of Psychology, 77,* 97-108. doi:10.1111/j.2044-8295.1986.tb01985.x

Watts, F. N., & Sharrock, R. (1984). Questionnaire dimensions of spider phobia. *Behaviour Research and Therapy, 22,* 575-580. doi: 10.1016/0005-7967(84)90061-5

Wechsler, D. (2001). *Wechsler Test of Adult Reading (WTAR).* San Antonio: The Psychological Corporation.

Wolpe, J. (1969). *The practice of behavior therapy*. New York: Pergamon Press.

Woodman, G. F. (2010). A brief introduction to the use of event-related potentials in studies of perception and attention. *Attention, Perception and Psychophysics, 72,* 2031-2046. doi:10.3758/APP.72.8.2031

Zhu, D. C., Zacks, R. T., & Slade, J. M. (2010). Brain activation during interference resolution in young and older adults: An fMRI study. *Neuroimage, 50*, 810-817. doi:10.1016/j.neuroimage.2009.12.087

Appendix A

Online Screening Questionnaire

Screening Questionnaire

Section 1 - Demographics

1. Age _____

2. Sex _____

3. Females only:

Are you currently on the contraceptive pill? **Yes / No**
Are you currently pregnant or breastfeeding? **Yes / No**
Is there any possibility that you could be pregnant? **Yes / No**

4. Is English your first language? Yes/no?

(if no please specify_____)

5. Are you left or right handed? Right [1] Left [2]

6. What grade of school did you complete?

Year_____

7. Have you completed any courses after school?

No……0

Yes, trade/technical…...1

Yes, university……2

Specify qualifications_____

8. Are you currently studying?

No……0

Yes, trade/technical…….1

Yes, university…….. 2

Specify _____

Section 3 – Health and Medical History

1. Have you ever suffered from any of the following:

Epilepsy **Yes** **No**

Severe head injury **Yes** **No**

Diabetes **Yes** **No**

Fits or convulsions (that were not related to a fever) **Yes** **No**

Loss of consciousness (greater than 2 minutes) **Yes** **No**

Concussion in last 6 weeks **Yes** **No**

Regular Giddiness **Yes** **No**

Heart condition or any other serious physical condition **Yes** **No**

Sleep disorder (or any major sleeping difficulties) **Yes** **No**

Visual problems (that are not fixed with glasses/contact lenses) **Yes** **No**

Hearing problems **Yes** **No**

If you answered yes to any of the questions above, please provide some extra information on the condition (and the length of time and severity.

2. Are you currently taking any prescribed medications? Yes / No

If yes, please specify:

.....
.....
.....

3. Do you have sensitive skin? Yes / No

(Skin preparation for EEG recording includes using alcohol wipes and exfoliant in order to get the best reading possible from electrodes, people with sensitive skin may find this irritating)

Section 4 – Mental health

1. Have you ever been diagnosed with a mental health condition? Yes / No

If yes, please provide some extra information (including the condition and time frame):

Section 5 – Substance use

The following questions are about your use of tobacco, alcohol and other substances

1. In the last 6 months, how often have you used tobacco/nicotine?

Never 0

Less than monthly 1

Monthly 2

Weekly 3

Daily or almost daily 4

2. In the last 6 months, how often have you used illicit drugs (e.g., cannabis, ecstasy, speed)?

Never 0

Less than monthly 1

Monthly 2

Weekly 3

Daily or almost daily 4

3. On how many occasions have you ever used illicit drugs?

None 0

1-5 1

5-10 2

10-15 3

More than 15 occasions 4

Appendix B

Ethics Approval Email

Removed for confidentiality purposes.

Appendix C

Participant Information Sheet and Consent Form

PARTICIPANT INFORMATION SHEET

Spider Fear, Brain Activity, and Attention

Invitation

You are invited to participate in a research study into the effects of spider fear on attention during the viewing of spider images. This is an Honours study being conducted by Monique Williams and Tess Nikitenko under the supervision of Dr Allison Matthews (Chief Investigator, School of Medicine, Psychology).

1. 'What is the purpose of this study?'

The purpose is to investigate brain processes involved in attentional processing among males and females with high and low spider fear.

2. 'Why have I been invited to participate in this study?'

You are eligible to participate in this study because you have an intense fear of spiders or that you have a relatively low of fear spiders.

4. 'What does this study involve?'

This study will require you to attend one session (approximately 2 hours) at the University of Tasmania. In this session you will complete some questionnaires relating to your fear of spiders. You will then complete some computer tasks where you will respond (using a button press) to particular aspects of visual stimuli presented on a computer screen. These stimuli may include pictures, letters or objects (and may include pictures of spiders). Your brain activity will be measured while you complete these tasks.

It is important that you understand that your involvement is this study is voluntary. While we would be pleased to have you participate, we respect your right to decline. There will be no consequences to you if you decide not to participate, and this will not affect your relationship with the University. If you decide to discontinue participation at any time, you may do so without providing an explanation. All information will be treated in a confidential manner, and your name will not be used in any publication arising out of the research. All of the research will be kept in a locked cabinet in the office of Dr Allison Matthews or on a secure server at the University of Tasmania.

5. Are there any possible benefits from participation in this study?

You may or may not experience anxiety during the course of the study. However, if you do, it is hoped that you will notice a reduction in your anxiety after a certain period of time. The results of this study will provide valuable information on the attentional processes involved in spider fear and will help us to further develop an online treatment program for people with phobias.

6. Are there any possible risks from participation in this study?

If you experience anxiety during the study, this may be unpleasant and include emotions of fear and worrying thoughts, wishing to avoid the situation, physical discomforts such as palpitations, sweating and over-breathing. The researchers will provide you with information for dealing with these symptoms if they unduly trouble you. However, if you find that you are becoming distressed or experience significantly elevated levels of anxiety you will be advised to receive support from a clinician or alternatively, we will arrange for you to see a counsellor at no expense to you..

There are no specific risks associated with the measurement of brain activity. However, if you have sensitive skin there is a small possibility of a slight skin reaction from electrode preparation materials. If you believe there is a chance that your skin may react you are advised to reconsider participation.

7. What if I have questions about this research?

If you would like to discuss any aspect of this study, or require further assistance with your fear of spiders after the study is completed, please feel free to contact Dr Allison Matthews on ph (03) 62267236, who would be happy to discuss any aspect of the research with you. Once we have analysed the information we will be putting a summary of our findings on the School of Psychology website for you to view. You are welcome to contact us at that time to discuss any issue relating to the research study.

This study has been approved by the Tasmanian Social Science Human Research Ethics Committee. If you have concerns or complaints about the conduct of this study should contact the Executive Officer of the HREC (Tasmania) Network on (03) 6226 7479 or email human.ethics@utas.edu.au. The Executive Officer is the person nominated to receive complaints from research participants. You will need to quote [H0011104].

Thank you for taking the time to consider this study. If you wish to take part in it, please sign the attached consent form. This information sheet is for you to keep.

CONSENT FORM

Spider Fear, Brain Activity, and Attention

1. I have read and understood the 'Information Sheet' for this project.
2. The nature and possible effects of the study have been explained to me.
3. I understand that the study involves attending one session (approx. 2 hours) at the University of Tasmania whereby I will complete some questionnaires and some computer based attention tasks. These tasks may involve responding to pictures (including spiders), letters, or objects and brain activity will be monitored throughout the process.
4. I understand that participation involves some risk of experiencing a heightened level of anxiety; however, the researcher will be present at all times, I will be given information on how to cope with anxiety, and I will be referred to a counsellor if need be. I understand that measurement of brain activity involves minimal risk, and slight skin irritation may occur if I have sensitive skin.
5. I understand that all research data will be securely stored on the University of Tasmania premises for ten years and will then be destroyed.
6. Any questions that I have asked have been answered to my satisfaction.
7. I agree that research data gathered from me for the study may be published provided that I cannot be identified as a participant.
8. I understand that the researchers will maintain my identity confidential and that any information I supply to the researcher(s) will be used only for the purposes of the research.
9. I agree to participate in this investigation and understand that I may withdraw at any time without any effect, and if I so wish, may request that any data I have supplied to date be withdrawn from the research.

Name of Participant: _____

Signature: _____ Date: _____

Statement by Investigator

☐ I have explained the project & the implications of participation in it to this volunteer and I believe that the consent is informed and that he/she understands the implications of participation

If the Investigator has not had an opportunity to talk to participants prior to them participating, the following must be ticked.

☐ The participant has received the Information Sheet where my details have been provided so participants have the opportunity to contact me prior to consenting to participate in this project.

Name of Investigator

Signature of Investigator

Appendix D

Experimental Questionnaire

Date ____/____/____

Participant ID _____

1. **Check that participant has abstained from alcohol for 24 hours and illicit drug use since completing the screening questionnaire**

3. **How many cups of coffee (or any other caffeinated drinks/products) have you consumed today? _____**

   If > 0. How many hours since your last caffeinated drink _____ hours

4. **Have you had any tobacco or nicotine products today? Yes / No**

   If yes, how many cigarettes (or nicotine products) have you had today? ____

   If yes, How many hours since your last cigarette (nicotine product) _____ hours

5. **Have you consumed any medications in the past week (or any prescribed medications since completing the screening questionnaire)?**

   If yes, please detail:

Medication	Number of occasions	Time since last used	Estimated dose

6. Approximately how many hours sleep did you have last night? ____

Karolinska sleepiness scale (participant can self-complete)

Please circle on the following scale of 1 to 9 how you feel **AT THE PRESENT MOMENT**:

1	2	3	4	5	6	7	8	9
Very alert		Alert – normal level		Neither alert nor sleepy		Sleepy – but no effort to stay awake		Very sleepy, great effort to stay awake, fighting

Appendix E

Participant Video Gaming Experience Questionnaire

Date: _____ Participant: _____

Video Gaming Experience Questionnaire

We are interested in how often you play video games, and may use this information to examine the effects of video game playing on visual attention and motor skills.

How often would you normally play video games? Please choose one response.

- ☐ Never play video games
- ☐ Rarely play video games (less than 2 hours a month)
- ☐ Occasionally play video games (between 30 minutes and 2 hours a week)
- ☐ Regularly play video games (between 2 hours and 5 hours a week)
- ☐ Often play video games (more than 5 hours a week)

Appendix F

Non-Significant and Non-Theoretically F-Statistics

Table F1

F Statistics for Non-significant and Non-theoretically Relevant Effects in Accuracy, Reaction Time and P1 Amplitude Analyses for Go Stimuli

Effect	*F*-test
Accuracy (% correct)	
Group	$F(1,28)=3.00$, $p=.094$, $\eta_p^2=.097$
Target image	$F(1,28)=3.42$, $p=.075$, $\eta_p^2=.109$
Group x Flanker type	$F(1,28)=1.14$, $p=.296$, $\eta_p^2=.039$
Group x Target image	$F(1,28)=.017$, $p=.896$, $\eta_p^2=.001$
Flanker type x Target image	$F(1,28)=1.92$, $p=.177$, $\eta_p^2=.064$
Group x Flanker type x Target image	$F(1,28)=1.03$, $p=.318$, $\eta_p^2=.036$
Reaction Time (ms)	
Group	$F(1,28)=2.26$, $p=.144$, $\eta_p^2=.075$
Group x Flanker type	$F(1,28)=.002$, $p=.967$, $\eta_p^2<.001$
Target image x Flanker type	$F(1,28)=0.92$, $p=.346$, $\eta_p^2=.032$
Pl amplitude	
Hemisphere	$F(1,28)=0.86$, $p=.363$, $\eta_p^2=.030$
Flanker type x Group	$F(1,28)=0.01$, $p=.921$, $\eta_p^2<.001$
Flanker type x Target image	$F(1,28)=0.01$, $p=.914$, $\eta_p^2<.001$
Flanker type x Hemisphere	$F(1,28)=0.07$, $p=.789$, $\eta_p^2=.003$
Target image x Hemisphere	$F(1,28)=0.03$, $p=.864$, $\eta_p^2=.001$
Group x Target image x Flanker type	$F(1,28)=0.34$, $p=.567$, $\eta_p^2=.012$
Group x Target image x Hemisphere	$F(1,28)=0.30$, $p=.590$, $\eta_p^2=.010$
Group x Flanker type x Hemisphere	$F(1,28)=0.47$, $p=.500$, $\eta_p^2=.016$
Target image x Flanker type x Hemisphere	$F(1,28)=2.57$, $p=.120$, $\eta_p^2=.084$
Target image x Flanker type x Hemisphere x Group	$F(1,28)=0.74$, $p=.398$, $\eta_p^2=.026$

Table F2

F Statistics for Non-significant and Non-theoretically Relevant Effects in Accuracy and N2 Amplitude Analyses for Incongruent Image Go and Nogo Stimuli

Effect	*F* Statistic
Accuracy (% correct)	
Group x Flanker image	$F(1,28)=0.11$, $p=.748$, $\eta_p^2=.004$
Group x Trial	$F(1,28)=2.19$, $p=.150$, $\eta_p^2=.072$
Flanker image x Trial	$F(1,28)=3.68$, $p=.065$, $\eta_p^2=.116$
Flanker image x Trial x Group	$F(1,28)=0.33$, $p=.570$, $\eta_p^2=.012$
Peak N2 Amplitude (μV)	
Group	$F(1,28)=0.71$, $p=.405$, $\eta_p^2=.025$
Flanker image	$F(1,28)=0.71$, $p=.405$, $\eta_p^2=.025$
Group x Trial	$F(1,28)=1.04$, $p=.318$, $\eta_p^2=.036$
Flanker image x Trial	$F(1,28)=0.02$, $p=.893$, $\eta_p^2=.001$